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TRANSACTIONS

OF THE

THIRTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

HELD AT

COLORADO SPRINGS, COLO.,

SEPTEMBER 13-18, 1926

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JOINT SESSIONS

TABLE OF CONTENTS

PAGE

President's Address.
E. C. ELLETT, M.D., Memphis, Tenn..... 17

"Hearing" by Touch. Demonstration of a Case.
PROF. ROBERT H. GAULT, Washington, D. C..... 25

The Use of Foreign Bodies in Eye Surgery.
H. GIFFORD, M.D., Omaha, Neb..... 35

Ophthalmic Plastic Surgery.
WALTER R. PARKER, M.D., Detroit, Mich..... 44

The Use of Foreign Bodies in Ear, Nose and Throat Surgery.
HARRY L. POLLOCK, M.D., Chicago, Ill..... 52

The Surgery of Senile Cataract.
MR. BASIL GRAVES, London, Eng..... 68

The Anomalous Forms of Tuberculosis.
GERALD B. WEBB, M.D., Colorado Springs, Colo. 90

The Use of Tuberculin in Ocular Tuberculosis.
WILLIAM C. FINNOFF, M.D., Denver, Colo..... 95

Tuberculosis of the Middle Ear and Mastoid.
FRANK R. SPENCER, M.D., Boulder, Colo..... 109

The Ophthalmologist and Oto-Laryngologist in Periodic Health Examinations.
E. H. CARY, M.D., Dallas, Tex..... 120

OPHTHALMOLOGIC SESSIONS

TABLE OF CONTENTS

PAGE

A Study of the Nutrition of the Crystalline Lens.
DANIEL B. KIRBY, M.D., New York City, N. Y... 137

Diagnosis and Treatment of Vertical Deviations of the Eyes.
RODERIC O'CONNOR, M.D., Oakland, Cal..... 155

Gastrointestinal Toxemia and Its Relation to Ocular Pathology.
FREDERICK F. TEAL, M.D., Lincoln, Neb..... 176

The Reduction of Glaucomatous Tension by Deep Iridectomy.
H. W. WOODRUFF, M.D., Joliet, Ill..... 186

The Oculocardiac Reflex.
HARRY S. GRADLE, M.D., Chicago, Ill..... 205

Social Service and Follow Up in Ophthalmology.
CONRAD BERENS, M.D., New York City, N.Y..... 226

Band Keratitis.
ARTHUR J. BEDELL, M.D., Albany, N. Y..... 243

Fracture Involving the Apex of the Orbit, With Little or No External Evidence of Trauma, Resulting in Blindness of One Eye.
EDWARD B. HECKEL, M.D., Pittsburgh, Pa..... 256

Cataracts: Operation for Congenital and Juvenile.
F. W. DEAN, M.D., Council Bluffs, Iowa..... 261

Melanotic Sarcoma of the Choroid. Report of Two Cases. Bibliography.
C. NORMAN HOWARD, M.D., Warsaw, Ind..... 271

Annular Malignant Melanoma (Ring Sarcoma) of the Iris.
WILLIAM R. MURRAY, M.D., Minneapolis, Minn. 296

OTO-LARYNGOLOGIC SESSIONS

TABLE OF CONTENTS

PAGE

Scarlet Fever Statistics.
ANNA B. LEFLER, M.D., Los Angeles, Cal..... 309

Further Observations of Mastoid Infections in Infants.
ARTHUR M. ALDEN, M.D., St. Louis, Mo..... 316

Minimizing the Terror of an Operation.
SECORD H. LARGE, M.D., Cleveland, Ohio..... 326

Local Anesthesia in External Operations on the Nasal Accessory Sinuses.
ROBIN HARRIS, M.D., and W. LIKELY SIMPSON, M.D., Memphis, Tenn..... 341

Allergy As Related to Otolaryngology.
W. W. DUKE, M.D., Kansas City, Mo..... 354

Faulty Nutrition As a Causative Factor in Infections of the Upper Respiratory Tract.
AMY L. DANIELS, Ph.D., Iowa City, Ia..... 367

Headaches—The Nasal Aspect.
ROSS HALL SKILLERN, M.D., Philadelphia, Pa... 378

Polypoid Disease of the Antrum.
EDWARD KING, M.D., Cincinnati, Ohio..... 388

Peroral Endoscopy in Its Relation to Otolaryngology, With Special Reference to Postoperative Complications.
GABRIEL TUCKER, M.D., Philadelphia, Pa..... 395

Herpes Zoster Oticus.
WILLIAM HARDIN SEARS, M.D., Huntingdon, Pa. 406

DEMONSTRATION OF INSTRUMENTS, ETC.

PAGE

A New Procedure in the Treatment of Chronic Maxillary Sinus Suppuration in Children.
ARTHUR M. ALDEN, M.D., St. Louis, Mo..... 432

Head Mirror.
H. B. LEMERE, M.D., Omaha, Neb..... 438

Photography of the Fundus.
ROBERT VON DER HEYDT, M.D., Chicago, Ill..... 439

Folding Beebe Loupe.
GORDON F. HARKNESS, M.D., Davenport, Ia..... 442

Irrigation and Drainage Tube.
GORDON F. HARKNESS, M.D., Davenport, Ia..... 444

An Ophthalmic Irrigating Basin.
HARRY S. GRADLE, M.D., Chicago, Ill..... 445

Bronchoscope Shield.
HOWARD L. STITT, M.D., Cincinnati, Ohio..... 446

A New Set of Test Letters for Near and for Distance Vision.
ALBERT C. SNELL, M.D., Rochester, N. Y..... 447

Necrology 450

Minutes 451

Alphabetic List of Members..... 481

Geographic List of Members..... 519

Index 565

TRANSACTIONS

OF THE

THIRTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

JOINT SESSIONS

PRESIDENT'S ADDRESS.

E. C. Ellett, M.D.

MEMPHIS, TENN.

May I first express my gratitude and appreciation to the members of the Academy for the honor of being elected to this office? It is no less a pleasure than it is a compliment to be intrusted with a share in its management, and in working for the realization of the ideals to which the Academy stands committed. As I have inherited this trust from a long line of distinguished predecessors, so I will yield it to others no less capable and worthy of your confidence than they, whose efforts will surely crystallize the many worthy purposes for which we are still striving.

The Academy observes at this time its 31st Annual meeting, having been founded under another name in 1895. From small beginnings it has grown steadily. In 1906, there were 529 members; in 1916, 1,157, and there are now on the roll 1,543. It has thus become the largest body of eye, ear, nose and throat men in the world, and with constantly increasing requirements for membership, it increases in usefulness, dignity and the possibilities for service. Besides the valuable contributions to the literature of the several specialties contained in the volumes of the Transactions, the imprint of its influence is seen in other fields as well. The Academy was a factor in the organization of the American Board of Ophthalmic Examinations, now completing its 10th year of activity. It participated in the organization of the similar Board for Oto-Laryngology, which is functioning in an efficient manner. It aided in the foundation of a department of ophthalmic and otolaryngic pathology in the Army Medical Museum, the value of which may be judged by the excellent exhibit seen at this meeting. It has founded and successfully conducted an intensive postgraduate course in the shape of the Section on Instruction, whose 6th session, to be held at the close of this meeting, is a monument to those who conceived the idea, and especially to those two who have served on the Committee

from the beginning, the Chairman, Dr. Gradle, and the Secretary, Dr. Wherry. The Academy lent its influence to the organization of an International Congress of Ophthalmology in this country in 1922. It is also fostering certain research, a report of which will be made at this meeting. It is not enough, however, that we shall have put these and other activities in motion, but we must separately and collectively stand behind them and see that they realize the purpose for which they were originated. The examining Boards in Ophthalmology and Oto-Laryngology have been greatly helped by the attitude of this and other societies in making the possession of the respective certificate a requisite for membership, and we should in our individual capacities do what we can to make these certificates actually mean something. Each of us should use his influence to see that in the matter of hospital and college appointments the possession of the Certificate should be made to count, if, indeed, the applicant should not be required to possess it or to apply for it immediately. The recently opened Eye and Ear Hospital in Memphis requires that all of the surgeons and associate surgeons shall have or shall acquire the certificate of one of the Boards, and promotion in the Staff is dependent on the possession of the certificate. In the employment of assistants or recommending them to others, the value of the certificate should be emphasized, till it will be incumbent on every one who poses as a specialist in ophthalmology or otolaryngology to have a certificate or explain why he has not. Examinations are held by each Board at every meeting of the Academy, as well as at other times, and Fellows should try to visit the examinations, to which they are welcome, and see for themselves that they are conducted in an earnest, fair manner, but thoroly, and that they constitute a reliable test of the candidate's fitness to practice his specialty.

There will be presented to you at the proper time a statement of the affairs of the Army Medical Museum as it affects the department of ophthalmic and otolaryngic pathology. Briefly, it is to the effect that the demands of the work sent in by us have so grown that it overtaxes the facilities of the regular personnel, and it will be advisable for the Academy and other interested organizations to lend financial assistance, at least to the extent of paying the salary of one technician. The Army Medical Museum will receive from you, as you

know, any specimen of any pathologic condition affecting the eye, ear, nose or throat, make the necessary sections and examination and send you a slide and a report without any expense to you. Too few of our members avail themselves of this service, but if they will do so, there will be built up in time a museum whose material will be available for study, and which will be of the greatest value.

Research is a hard problem. It may surprise you to know that the difficulty in regard to research is not to finance it, but to find the men capable and willing to do it. The cooperation of every Fellow is desired, that the Committee may know of any one with a problem, and the ability and willingness to work on it.

A recent feature of the meetings, and one from which great things are expected, is the suggestion box which you will find at the door. It is earnestly requested that every one who has a suggestion about anything that will be a help to the Academy or its program, or any of its activities, will give the Council the benefit of that suggestion, with the assurance that it will receive the most earnest consideration. This Academy is meant to be a democratic body, and its affairs are the affairs of every member of it, and this suggestion box is the best way for any Fellow to bring his views to the notice of the Council with the least trouble to himself.

An important matter that will be presented by the Council for your consideration and action is one of reorganization of the administrative part of the Academy. As conducted at present, by one Secretary, the many things brought to him for consideration, adjustment and action are so numerous and varied, that it has long been thought by those familiar with the situation, that some sort of reorganization of that part of the Academy was necessary, and it has been the subject of earnest consideration by the Council and other members of the Academy. The plan that will probably be presented for your action contemplates the creation of the office of General Secretary, or Secretary-General, or whatever you choose to call him, and three Assistant Secretaries, one for Ophthalmology, one for Oto-Laryngology and one for the Section on Instruction, thru whom all the activities of the Academy may be closely coordinated. This sketchy outline of the plan is to acquaint you with its general features, and enable you to give the matter some thought before it is presented to you for

your endorsement. Some change of the sort is necessary, as the business of the Academy is growing and is now so great, that the machinery that sufficed for its transaction in the past is no longer adequate. No other change is thought to be advisable, but any sugestions in regard to this important question will be very gladly received.

There are certain large questions before the profession at this time, with which the members of this Academy are more or less concerned, and to some of which your attention will be called during this meeting. Probably the biggest thing right now is the campaign for periodic health examinations, which is receiving so much support from the A. M. A. When we consider the very intimate and important relations which the organs in which we are especially interested bear to the general health of the individual, it becomes apparent that no survey of a person from the standpoint of his general health and prospect of life can be complete unless it takes into consideration such questions as the state of his retinal vessels, the question of chronic aural suppuration, and the state of his tonsils, accessory nasal sinuses and hearing. It is our duty as well as our right to call attention to these things, so that we may play a part in this big movement, and that we shall be considered and respected for the valuable information that we can add to the survey of a man's physical condition. These same things should also be called to the attention of life insurance companies, to whom they are valuable and usually neglected sources of information regarding a person's insurability and expectancy of life.

As in other years, we have this year the privilege of listening to a distinguished visitor from another country, who makes a long journey in order to be with us. Especially identified with one of the recent developments in ophthalmology in which he has done much pioneer work, his contribution to the program of this meeting will show us his interest and acquaintance with other parts of the subject as well. The Section on Instruction is also fortunate in having him as one of the contributors, and you will there have an opportunity to hear from Mr. Graves some of the more recent developments of the slit lamp and their application to questions of diagnosis and treatment.

There are several things that are constantly coming before us for consideration and possible settlement, and one of the

important ones that concerns many members of this Academy is that of the activities of the refracting optician, under one of the many names by which he seeks to be called. There are two phases to this question. One is the relation of the optician to the public in his capacity as an adviser in regard to the health of their eyes, and the other is that of our relation to him as a seller of glasses and sometime dispenser of our prescriptions. As regards the first phase, we are all of one mind, but whatever we may think about the advisability of men without proper qualifications fitting glasses, it is not going to be settled by what we think, but by the final test of the ability of such persons to render an adequate and satisfactory service. The activities of the members of this calling or "profession," as they want to be termed, were fully considered in a paper by Dr. J. M. Patton at the A. M. A. in 1924, and the extent to which they had gone in their efforts to secure legislation favorable to the placing of the whole matter of fitting glasses in their hands was something that not many of us realized till the reading of that paper. Their chief aim seems to be to have the right to fit glasses taken even from the medical profession unless the physician takes a course of study prescribed by them. The basis of their argument is that no such preparation is given in the regular medical course, an argument that might appeal to one at first glance, tho it would soon occur to one to inquire if this was any more of a drawback than to confer this right on one qualified only by the possession of this information, and uninformed as to the anatomy, physiology, chemical and other vital properties of the bodies of men, of which the eye is a small but integral part. We are awake, and thru our examining boards, special societies and other agencies, we are striving to see, recognize and correct our own shortcomings, as has always been the ideal of the medical profession. Our aim should be twofold, to remedy our own defects and to make the medical specialist in Ophthalmology the best qualified in fact to render service and to give advice in all matters of ocular health, disease and defects, and also to educate the public to know the facts, so that they may judge as to who is best fitted to be intrusted with the great responsibility of the people's eyesight.

The second phase of this question has to do with the sale of merchandise, namely glasses. The manner in which this is and has been handled in many places, especially the towns

and smaller cities, in which the oculist either furnishes or checks and stands responsible for the fitting of the glasses, is known to most of you, and you also know that the problem is essentially different from that in the larger cities, where the services of a competent prescription optician are and have always been available. Two years ago, this question came up for consideration at the A. M. A., and resulted in the appointment of a committee and the passage of certain resolutions, which, however, do not seem to have effected any real progress toward a solution of this question. Now it seems that some sort of a suggestion has come from the opticians to the effect that they would like to meet representatives of the medical profession for a discussion of some phases of this subject, and the Section on Ophthalmology of the A. M. A. appointed a committee of one on Optometry, and asked the A. O. S. and this body to appoint similar committees that the three might act together. The A. O. S. thought the matter of sufficient importance to suggest that the committees consist of three members from each society, and this will be presented to you at the proper time. We must be very careful that no unwarranted interpretation is put on the participation of the medical profession in such a conference, and the committee from this society should be so instructed. If the committee from this Academy is to have the same title as that appointed by the Section of Ophthalmology of the A. M. A., namely a Committee on Optometry, it might be well to have them also consider the matter of the interest of the oculist in the dispensing of glasses, in order that some sort of an understanding of the question should be arrived at that will represent the opinion and practice of those who are concerned with this matter, and a conclusion announced which will receive the support of all who are interested.

Last year, the experiment was made of devoting an evening to a business session. In that instance the experiment was not a success, partly owing to the attractions of the white lights and diversions of a great city for the members from the provinces. In view of the fact that no such counterattractions will contend for the interest of the members at this meeting, the program committee has decided to try this again, and the evening of the first day has been set aside for a business meeting. It is hoped that this meeting will be well attended, and that every one will feel free to express his opinion on all

the matters that will be presented for consideration. An impression exists in some minds that the Academy is run by a small group of members. It is! Every organization is run by such a group, which consists of a relatively small number of men who are active, who have the interests of the organization at heart, who attend regularly all the meetings, who do cheerfully and well all that they are called on to do, and who contribute more than their share to the work of the body. But such a group is not a close corporation. It is open to any man who has the will and the energy to do something, and one purpose of devoting a whole evening to the business of the Academy is that every man will have the opportunity to learn all he wants to know about the Academy's affairs, and to give his views, advice and help in every matter that concerns the Academy and its members. So the program committee requests that you will all come to this meeting, which will be made interesting, and if you do not come and the burden falls, as it has fallen before, on a few men to do the work, it will be in a way a failure of the ideal which should prevail in this Academy, to make it the big democratic body of eye, ear, nose and throat specialists, and to truly represent them in every way. The interests of this Academy are very large, as they should be. It is not only the largest but the wealthiest society of its kind in the world, and with size and wealth and power and influence there goes a great and serious responsibility, that all of these resources and strength should be used for the very best interests, not only of the branch of the medical profession which we represent, but even more for the interests of the great mass of people over whose senses we assume a certain guardianship, and who look to us for the proper discharge of the duties of the guardianship, not only in the letter but in the spirit. Neither as individuals nor as a body must we be found wanting.

Death has dealt harshly with this society since our last meeting. Never before have we lost so many valuable members in the same short space of time. Not only is their loss greatly felt, but it must serve to remind us that such things are to be expected, and the older ones of us are all to be called on at a more or less short interval to pay their final debt to Nature. Speaking as one of the older ones, then, let me say to the younger men and women who are coming on to take our places, that there must never be any let up in our strife

with the ills which we seek to overcome. This society must close ranks and continue to advance against an enemy who never ceases to fight. In the name of those who have passed, as well as in the name of those who may not be in the fight much longer, may I quote to the younger men the words of a great physician:

> "Take up our quarrel with the foe.
> To you from failing hands we throw
> The torch. Be yours to hold it high.
> If ye break faith with us who die,
> We shall not sleep."

"HEARING" BY TOUCH: DEMONSTRATION OF A CASE.

PROF. ROBERT H. GAULT.

WASHINGTON, D. C.

Undoubtedly there are many in the audience who have not yet had a good opportunity to be acquainted, to any degree of detail, with the work I am doing. Before I go on, therefore, to demonstrate a case, permit me to touch the high points in the enterprise before me so far as it has proceeded, and to get the whole work in the proper perspective.

During the past four years—more especially during the last two—I have been working upon a method whereby fingers are made to substitute for ears; a method by which a speaker's vocal vibrations are instrumentally communicated to the fingers or palm of a subject in such a manner, that the words spoken can be felt by a considerable group of persons simultaneously, whether they are near to the speaker or far away from him and out of his sight completely. But I have sought not only to have the words felt; the subject must be able to notice distinctions among words as felt, so that he may learn ultimately to associate feels with meanings, and to understand speech thru the medium of touch impressions.

In the next place, I have been developing a method whereby the feel of words and their elements may be used to aid the mute in getting control of his vocal expression. The mute is as he is in respect to speech because he cannot hear even his own voice. He is, therefore, unable to make correction of his speech. But when he can feel his own voice and mine in succession upon the same word, he shortly has some cues to guide him in the act of speech.

This is a broad, fundamental psychologic problem. More accurately, it lies upon the borders of three sciences: Psychology, Physiology and Physics. Like every other fundamental problem, it radiates in many directions. It runs into numerous psychologic problems of a very technical nature—the problem of meaning for example. It takes off into the

science of phonetics, and it offers a most unique and valuable starting point for the scientific study of the genesis of a language from its very beginning. Here is a handle that men of science in future will make much of, those who are interested in the development of language.

What I have said will, I hope, set my own work in the proper perspective. The fundamental problem runs into the practical ones that I have already mentioned relating to the deaf, enabling them to receive and interpret speech and to control their own vocal expression. These ends cannot be attained by one leap from the seafoam. Our technic can not be made perfect at a stroke. It must be preceded by a great deal of patient investigation into the backgrounds.

For those of you who are not already well acquainted with the enterprise, I want to mention the high lights among our accomplishments so far.

Last May, two of our subjects attained the capacity to interpret a story couched in colloquial English, when they had nothing to go by but its feel communicated thru the receiver of our apparatus to the fingers of the subject. In two years, they had an aggregate of 290 and 280 hours of training in the laboratory. Others of our group picked up words and isolated sentences, but failed to report the stories as connected discourse.

This accomplishment was preceded in the course of two years by a great deal of drill upon a large variety of exercises.

There have been many byproducts of all the work leading up to the accomplishment I have mentioned—byproducts that, for the most part, were unforeseen at the outset.

Our subjects have acquired a familiarity with the movement of speech that they never had before: they know its accent, emphasis, rhythm and tempo. These are qualities that can hardly, if at all, be got by practice in reading lips. As a consequence, these people enjoy speech as they could not otherwise. They have been shot thru with a language sense as you and I have been; only we have acquired it thru the medium of our ears—they thru their fingers. It is highly probably that they are well on the way toward feeling the lilt of speech that you and I experience. They are acquiring the same motor or kinesthetic associations with language that we have made.

All this is reflecting itself in our subjects' silent reading. They could in May mark a page in an unfamiliar book to indicate fairly well how a good reader would read it. In this respect, they were 28% superior to subjects of corresponding age and school experience, but who had never been within our laboratory till they were brought there on a certain day to undergo a test upon this point.

It is a fair hypothesis that familiarity with the movement of speech, however our people acquire it, will make them the better unaided lip readers in the future. It will require a long time and patient work to test this hypothesis. But there is good psychology to support it, and certain experiences that you and I have in silent reading support it also. But these are technical points into which I shall not enter now. It has encouraged me to find that some competent teachers of the deaf support the view I have taken.

What I have just now been saying will prepare you for my assertion that our cases enjoy verses as they are felt, and that they have their preferences amongst verses. Laboratory tests confirm the assertion. But their preference is not always confined to a certain rhythm of verse. Of two verses in the same rhythm, one containing predominantly the long or broad vowel and diphthongal qualities and the other the short qualities, the former is almost consistently preferred.

This observation has led me into preliminary experiments upon the reception and enjoyment of music. The results have been sufficiently promising to induce me to follow the investigation further.

But the result that suggests immediate practical value of large scope, I have yet to mention.

Let our subjects simultaneously see my face and feel my words whilst I speak; they can interpret my speech from 30% to more than 100% better than they can by observing my face alone. This has been demonstrated in our laboratory five times. The last demonstration was made by a skeptic who became convinced.

A result of this nature is assured in advance by two considerations:

Homophonous words like "aim" and "ape" that can not be distinguished by the lip reader, can be discriminated by their feel with but few exceptions at most. There are upwards of 2,000 such words in the English language.

In the second place, one can feel the movement of speech much more distinctly than one can see it, and this movement contributes toward the understanding of speech.

The superiority of lip-touch reading over lip reading gives assurance of a means whereby we may greatly speed up instruction in the schools and at the same time, I believe, enhance the pupil's ability to read lips without instrumental aid when he is outside the school and after school days are over.

Having reported this very brief summary of what has been accomplished, I shall show a few slides to illustrate our method and to afford a graphic representation of certain results.

Subjects and Hours.

The subjects or cases who have served in these experiments are deaf college students, aged from 17 to 28. For the most part, they are congenitally deaf or became deaf in very early life, before much language had developed. They range from freshmen to seniors in college. Three of them have long since left school and one of these is an instructor in Gallaudet College. The young woman who is here this evening is congenitally deaf. The audiometric test gives a zero result for each ear.

Within the year that closed last June, we worked with 18 cases that fall within the age group I have mentioned, and four seven year olds who were having their first school experience.

Of the 18, there were 10 who began their laboratory work about February first. Their aggregate number of hours spent in laboratory drill up to June ranged from 25 to 58 hours. The aggregate is made up of two 40 minute periods, daily for five days a week.

As to the remaining eight, their periods of drill aggregate from 110 hours to 290 hours. The young woman here has aggregated 110 hours. She first came into my laboratory on Oct. 16, 1925. All the others, aggregating up to 290 hours, have been in the laboratory since Oct. 8, 1924. Two of them, whose aggregates are 280 and 290 hours respectively, were able in May to report a story, tho they had no criteria to guide them but the feel of successive words and sentences upon their fingers. In other respects they are correspondingly proficient.

Naturally, I wished to bring one of them here to demonstrate this evening. Last June she agreed to come and was most enthusiastic over the prospect. Unfortunately, at the eleventh hour, she sent me word that she cannot be here on account of the very serious illness of her mother. I cast about in turn for three others. Two of them I could not locate, and the third had just taken a position from which she could not be released to make the journey to Colorado. I then wired Miss Dorothy Clark, and received her acceptance but fifteen minutes before I left Washington. And here she is.

Miss Clark is congenitally deaf. The audiometer makes her zero in each ear. She is a junior in Gallaudet College. Before she went to college, she was for 12 years a pupil in the State School in Colorado Springs. She began practice in our laboratory on Oct. 16, 1925, and has aggregated 110 hours of laboratory training. Seventy-three of these hours were before Jan. 1, 1926, and 37 since that date. Her college schedule made it impossible for her to be more than an intermittent attendant since Jan. 1. For these reasons, it is evident to you that I am not showing you tonight my "star pupil." Miss Clark has done extraordinarily well in the time she has spent in our laboratory, but her aggregate time has been but little more than one-third of the time several other subjects have given to the work.

You must bear it in mind that what you see her do tonight is done at the end of a long summer's vacation of no practice, during which she has had an unparalleled opportunity to forget. Since she arrived here the other day, we have given her but three periods of drill, aggregating about one hour.*

The instruments we have been using in these experiments have been lent to me by the Bell Telephone Laboratory in New York City. And that reminds me to say that we experimenters have no instruments to sell, and that we will not have them for sale. It is hardly necessary for me to say this to you physicians, but laymen sometimes appear to think that

*In the demonstration that followed, Miss Clark first recognized three out of ten vowel and diphthongal qualities by their tactual characters alone; in the second attempt she recognized only three of ten. She was plainly confused by the unusual situation. In the third trial, she identified eight out of ten. The ten qualities were a, e, i, o, u, oi, ou, er (middle western) oo, aw.

In the next place, she attempted to recognize colloquial sentences, each one of which might have been any one of twenty. In the first trial, she recognized only three; in the second, seven; and in the third, nine out of ten; or 6, 14 and 18 out of 20.

I am in the business (or will be) of making instruments to sell. I am an experimenter. Others must make the implements. I am furthermore in the position of the doctor who has discovered a specific. The ethics of the profession requires that he gives his knowledge to the public.

Further, speaking of instruments for our use in these experiments, I am glad to say that the Bell Telephone Laboratory is continuing its cooperation in a very practical way and at considerable expense. I expect, in October, to be equipped with new receivers that will be probably five times as efficient as the one we are using tonight.

People have been asking me what I foresee as a consequence of the experiments we are conducting. I believe we may fairly anticipate the day when schools for the deaf will have a transmitter analogous to this one in every recitation room, that at every desk occupied by a deaf pupil (or upon the pupil's body) will be an improved receiver to be used at the desk or carried to the blackboard to be plugged in there; that communication from teacher to pupil will be greatly facilitated thereby and instruction will be speeded. All of this will be accomplished without taking the deaf away from the language as it is spoken by persons of normal equipment. A further consequence will be found in the facilitation of silent reading, the improvement of vocal expression and even in the reception of music.

But this is not the whole vision. It is not improbable that an apt learner, after two or three years of special instruction to the end of developing a language sense thru the medium of touch, may take his place in the regular public school and compete with others there successfully. This assumes only that the regular school will equip itself with the necessary apparatus.

DISCUSSION.

Dr. Robert Sonnenschein, Chicago: It is difficult to properly discuss the splendid and very instructive demonstration of Professor Gault's for at least three reasons; One, because it is so comprehensive; two, because I had no opportunity of reading the paper in advance, and three, last but not least, because I have had no direct experience with the method.

It very often happens that certain principles which apply in one branch of medicine or other sciences find application in one way or another in other phases of the subject. In the proper functional testing of hearing, if the whispered or conversation voice is used, it is necessary that the individual not only have his eyes averted from the speaker, and the opposite ear closed, but that he be cautioned not to allow any part of his body, such as his elbow, to come in contact with the wall, doors, or other parts of the room in which the examination is being made. The reason for this is, that the sound might be conveyed to the head and thus to the inner ear by bone conduction and thereby a part of the test vitiated, in that one could not tell whether the hearing was entirely by air or not. We realize, of course, that whenever sound is received by the head, some of it is conducted by air and some by bone conduction. In those cases in which there is no direct contact of the sounding body with the head, we hear most of the sound waves by air conduction; but when there is contact with the head or other part of the body, the bone conduction carries most of the sound waves. Professor Gault has very ingeniously utilized this latter principle in his work, and I can readily see that there are great possibilities opened by this new avenue of research.

With Professor Gault's method, it is probably mainly by the sense of touch that sounds are appreciated and not by the sense of hearing. Pohlman showed with the socalled "bone knocker," that the deaf did not hear by way of the bones of the head as well as they did by way of the fingers or shins. In other words, we probably have here a substitution of the sense of touch for that of hearing.

Kranz states that "the patients learn to appreciate the sequences of peaks of vibration, and not the individual vibrations themselves. The ears are capable of hearing sounds of different frequencies from about 16 to 20,000 double vibrations per second. The amplitude of a sound of 2,000 double vibrations, which the ear is just capable of perceiving, is about one one-billionth of an inch, much smaller than any movement which can be detected by the sense of touch. Changes of ten percent in the intensity of ordinary loudness can be noticed by the ear, as can also changes of about one percent in the pitch. The sense of touch will detect changes in pressure of about twenty-five percent." He states that probably the differentiations made by the tactile sense in Professor Gault's work are those of rather gross changes in amplitude. Thus if a person had his fingers on a telephone receiver which was being activated by a 500 cycle current, it does not seem probable that he will follow the position of the diaphragm's excursions occurring in one five-hundredth of a second; but what will probably be detected will be changes in what may be called the "envelope of the motion," that is, the variations in the maximum positions of deflection of the diaphragm, which will probably occupy a time period of about one second rather than one five-hundredth of a second. Sentences would

probably be differentiated by the sequences of changes of amplitude, and since the sentence has a longer sequence than a word, the possibilities of variation are greater, and so it would be much easier to distinguish between sentences than between words. Kranz also believes that "further refinement of Professor Gault's apparatus would give a spatial differentiation of received frequencies. If four receivers were used, with the resonant frequencies of the diaphragm suitably separated, and a finger held on each of the diaphragms, one would obtain the spatial differentiation of the frequencies, which in combination with the amplitude discriminations now being used, should aid greatly in distinguishing various sounds or sound combinations. This would accomplish with perhaps even better control what Dr. Goldstein does in having children place the fingers on a membrane stretched over the end of a megaphone into which he is speaking."

As previously mentioned, I have had no personal experience with the procedure under discussion and, therefore, am not in a position to judge its practical value. The important factor seems to me to be the manner or degree in which it will aid the deaf in appreciating what is going on about them. The use of lip reading is of tremendous aid to these unfortunates, and it is something which can be acquired by most of them after a reasonable length of time and does not require the use of apparatus, and so forth. Whether Professor Gault's method (involving as it does the use of appliances) will be of material assistance to the hard of hearing from a practical standpoint, it seems to me it is too early to state. Whether it later demonstrates this value or not, he deserves the fullest measure of credit and thanks for the very excellent research along this auditory-psychologic line (if I may use such a term) and for his splendid presentation of the subject today and in his previous papers on the subject.

Dr. John F. Barnhill, Indianapolis: It is a matter of common observation that those who have been deprived of one sense acquire sharpening of other senses more readily than those who have all their senses, and that the degree of acquirement and the rapidity of that acquirement is rather amazing in those who are intelligently directed.

We have seen the demonstration of Dr. Goldstein. I well remember when the children stood with their hands upon the back of the piano, some one struck the keys and the children immediately sang the notes as played. In other words, they caught from the boards of that piano the key to the music, the music itself, and carried it on. It seems to me the principle is much the same as that in the demonstration we have just witnessed. I do not know that this is true, but that is the way it strikes me. It is a question of amplitude, and this young lady learns to recognize and to interpret the feeling that comes to her fingers, just as the children in Dr. Goldstein's classes learn to interpret music thru the vibrating body of the piano.

As a matter of practical observation, my thought as this demonstration was going on was that there is not the slightest deception about this telephone reading on the part of the young lady. I can, however, understand that of the twenty sentences before her, she would learn that there were five words in one sentence, ten in another, and etc.; knowing this much, she might guess something of the nature of the amplitude of each word as it comes thru, and this might help her to interpret. Take the

sentence, "She coughed all night." She has learned there is such a sentence and the number of words. I wonder if she might not be able to read the words without actually feeling them. In teaching her, no doubt these words have been given to her over and over again, and I wonder if the words were given to her in a different order, say, "night," "all," "she," "coughed"—mixing the words,—whether or not she would be able to interpret the word "night," "cough" or "all" if it came over the wire out of its regular order in the sentence.

Another thought that came to me, was whether or not it is wise to mix methods of teaching the deaf, to take away teaching by lip reading, or try to teach both methods. The professor has not said whether or not he advocates teaching both methods. My thought is, would it not be better to put the same intensity of thought and scientific work on lip reading alone, which requires no machinery and can be used by persons in the street as well as in the lecture or music hall, or wherever the pupil might be, for, when able to read the lips, pupils may converse, may ask questions on the street, which would be of great benefit. However, I can also see the benefit which the professor emphasized towards the close, namely, that thru this phone method, the rhythm and the accent of words and sentences can be distinguished. That, certainly, would be an enormous pleasure and might be worth all the efforts that could be put into it.

The title of the address was "Hearing Thru the Skin." The professor, of course, does not intend to convey the notion that it is actually hearing, but that it is a substitute for hearing, that is, interpreting thru the skin. It is a splendid demonstration, and I am greatly pleased to have been present to witness it.

Professor Robert H. Gault, Washington, D. C. (closing): Far be it from me to urge that these people are hearing thru their skin. That is utterly impossible. The use of the term "hearing" in this connection is figurative. My cases are feeling speech and they are learning to interpret what they feel. Their process of learning is probably the same that you and I had to go thru when we were learning to interpret certain bombardments of air particles against our auditory apparatus. The bombardments we got in the course of our learning are brought to the skin of these people, and they learn to interpret the feel of them. They are not hearing; they are no nearer hearing today than when we began to work with them.

The doctor questioned whether it is wise to get away from lip reading. We are not trying to do that. My thought, and I am pretty thoroly convinced, is that we may be helping the process of lip reading. I know very little about teaching the deaf, but I have been told by many experienced teachers of the deaf that it is extremely difficult for deaf people, observing the faces of speakers, to become acquainted with what we have called the movement of speech, accent, emphasis, rhythm, tempo—the varying tempo of speech. These are characteristics of speech that we can get over thru the sense of touch. Our subjects can feel accent and emphasis. You and I, in the course of the years of our life, have got into the way of associating movement of speech with its meaning. If I were to speak to you in a monotone, you would find even greater difficulty than usual in knowing what I mean. The rhythm, the movement, of my speech helps you to understand what I mean, and helps me to understand you. We feel that the young people who have been following the movement of

speech and accurately associating that movement with its meaning, do better lip reading than they could have done had they not had experience with the tactile apparatus, in school or elsewhere. There is enough good psychology back of this to lend a deal of credence to it, and I have been much interested recently in finding that some experienced teachers of the deaf have no doubt that the thing will work out in that way. I am not saying it will, but I believe it will do so.

One of the doctors spoke of the possible advantage of distributing the impressions of speech among several areas upon the skin. That idea has occurred to me, and it is being worked out by the Bell Telephone Laboratory for my use. They are making an apparatus that will stimulate the hand of the subject, not only at one point as in this instance tonight, but it will stimulate the tip of every finger and the thumb. That is, the whole range of frequency of vibrations employed in speech will be broken up into five parts or segments, and for each segment there is a unit in the receiver that the subject holds in his hand. The receiver we are using tonight has but one unit and it operates, of course, upon but one very small skin area on the tip of a finger that has been resting lightly upon a vibrating diaphragm. With five such units working upon as many different areas, reception is bound to be greatly improved. One of these units will carry a block of the highest frequencies used in speech, another the lowest and others will carry the remaining intermediate blocks. The word "a" will then be felt, not at one point alone, but at two or more. The variety of localizations of stimulation that ensues from the use of such an instrument will considerably multiply the opportunities of the learner to distinguish words and sentences and to become acquainted with the movement of speech.

By the same token, it should afford a distinct advantage as furnishing cues to aid the semimute to get control of his vocal expression.

(On motion, duly seconded, the Academy extended to Professor Gault a vote of thanks for his very splendid presentation. Motion carried by rising vote.)

THE USE OF FOREIGN BODIES IN EYE SURGERY.

H. GIFFORD, M.D.

OMAHA, NEB.

In considering this subject, which has been assigned to me by your Committee, I find that perhaps the most important use of foreign bodies in ophthalmology is that made in the attempt to improve the appearance of artificial eyes. Altho a list of the different substances which have been employed to this end would fill a small book, they may be summarized by dividing them into inorganic substances such as glass, silver, platinum and gold; organic or hetero- or homografts like bone, cartilage and sponge; and autografts, such as fat from the patient. All of these substances have been used indiscriminately for the Mules' or the Frost operation, that is, for the improvement of an evisceration stump or of that left by enucleation.

Probably three-fourths or even a larger proportion of these implants have been made of glass. Platinum, or the gold balls recommended by Fox,[1] are the least subject to deterioration, but perhaps on account of the subconscious thriftiness of the race, very few practitioners seem to use them. Altho glass, especially if not lead free, is known to undergo changes in contact with the tissues of the body, this change occurs so slowly that it very seldom causes any inconvenience; or if it does, it occurs so long after the original operation that the operator does not hear of it. In fact, he is sometimes dead, as is shown by a patient who came to me last June, on account of severe attacks of pain and edema in the stump of an eye, which he said had been removed in 1892 by an oculist who died some ten years ago. These attacks, of which four had occurred since February, were fugitive but extremely painful, and from the history there was some question as to whether he might not have angioneurotic edema. The patient did not know that his stump contained any glass, but investigation revealed a good Mules' stump containing a broken glass ball, much of which was so thin as to be easily bent. It was found to contain lead.

This incident, however, can hardly be considered a great discredit to the use of glass, since the ball had allowed the patient to wear an extra-good-looking glass eye thru thirty-four of his most sensitive years; and if he had desired it, another ball could have been put in. Doubtless, if a small fraction of the trouble used to give us the doubtful benefit of a new composition for spectacle glasses were to be employed on this subject, a glass ball could be made which would remain in the orbit unchanged for a lifetime at least; but any old glass seems to do so well, and experiments in this line would take such an unconscionable time for verification, that such a ball will probably remain a pious wish. It also seems probable that some alloy or treatment of silver could be found which would make it 100% resistant. Gold plated balls are not always reliable.

The introduction of an artificial vitreous into the evisceration stump was independently suggested by Kuhnt[2] shortly after Mules' operation was first performed. He used silver balls, however, which time has shown to disintegrate after some years. Aluminum, as suggested by Bryant,[3] had a meteoric career. These aluminum balls were light and cheap, but the aluminum proved much more unstable than silver. None of the numerous other substitutes for glass balls has had much vogue. Paraffin, perhaps, has been used more than any of them, and it is unobjectionable so far as I know. Whether, with the lapse of time, the paraffin balls show any disintegration or any important change of form or location—that is whether they stand the test of time as well as the glass balls—remains, I think, to be determined.

Of the heterografts, Magitot's[4] formalinized calves' cartilage balls, like every other novelty, have had some advocates; but they are not so easy to obtain as the glass ones, and it remains to be seen whether they have any real advantage over the latter. The same may be said of the heads of metacarpal bones and other bone balls, including the burned out ones of Schmidt.[5]

Some years ago, after having a case of sympathetic ophthalmia following a Mules' operation, the writer, on reviewing the available statistics, came to the conclusion that there was a somewhat greater risk of sympathetic ophthalmia from either a Mules' or a Frost operation, than from an ordinary evisceration or enucleation; and since that time I have used these operations only at the express wish of the

patient, after having my belief in the slightly greater danger explained to him. I have always felt, however, that the theoretic objection to such foreign bodies as glass and metal balls did not apply so strongly to the patient's own fat, as first recommended by Barraquer.[6] The cosmetic results obtained by its use, however, are not so good as those obtained by the more resistant substances, and I have had it in mind to try to improve the viability and permanent size of these autografts by including with the piece of fat the skin covering it, after having shaved off the epidermal layer. It seems to me that, especially in the cavities left by enucleation, there is some chance for a thick skin graft actually to live.

The main desiderata in an implant are innocuousness and permanence, and it is the attempt to reach the ideal in these particulars, which has led to the neverending stream of suggestions as to the materials to be used. But even if the ideal were attained in this direction, another difficulty would still remain to be overcome: namely, the tendency for the contracting tissues to extrude whatever form of implant may be used. To meet this, two lines of defense have been followed; one consisting of various measures for strengthening the anterior wall of the implant cavity; the other, the use of implants of such shape that they will tend to be retained by the tissues rather than to be extruded. Where the implantation has been done after an evisceration, the most important modification of the ordinary incision is that of Schmidt,[7] who, after excising the cornea, makes oblique incisions, so as to form four scleral flaps. The horizontal flaps are united in the vertical line, then the vertical flaps are united outside the horizontal ones, thus giving a double scleral wall in front of the cavity. I have tried this a few times in former years, with apparently good results. The objection to this is that it materially reduces the size of the implant cavity. On the other hand, a negative plan for increasing the strength of the wall is to avoid, as much as possible, any decrease in the size of the cavity, so that with a given sized ball, the scleral wound will have a longer time to become firmly united, before meeting the inevitable strain occasioned by the contraction of the sclera. This I have accomplished by leaving the cornea intact,[8] making a long incision for the evisceration, one-fourth inch or more above the cornea, after dissecting back the conjunctiva all around the cornea. The scleral wound is then closed with a carefully

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placed continuous suture of fine black silk, preferably including only the outer two-thirds of the sclera, and the conjunctiva is drawn over the cornea by a pursestring suture. This leaves no possible avenue for germs from the conjunctival sac to reach the suture or the scleral wound, and it permits the operator to use a larger sized ball than otherwise would be possible. The cornea, which is gradually exposed when the pursestring suture is removed, thickens, becomes opaque and loses its sensitiveness. The scleral suture seldom or never produces any irritation; but if it should seem desirable to remove it some months later, this can easily be done thru a few snips in the conjunctiva.

Theoretically, there is something to be said both for and against the practice of Huizinga,[9] in excising a piece of the sclera and of the optic nerve in evisceration. This makes a break in some of the natural lymph channels connecting the two eyes; but whether these channels may not reform, and on account of the hole in the back of the sclera, serve even better as a track for germs left in the cavity or in the substance of the sclera, is not certain. It may further be true, that even if the lymph channels of the central end of the optic nerve and its sheath are permanently closed by the resection, other channels thru the orbital tissue and the sphenoidal fissure may, on account of the hole in the sclera, afford an easier passage from the stump to the second eye.

The attempt to favor the retention of the implant by the ingrowth of tissue into it, has taken the form, on the one hand, of using balls of sponge, catgut, silk and other more or less permeable or absorbable substances. I have had no experience with these, and have seen no nonpartisan reports on the results of their use. In attempting to favor this ingrowth by the shape of the implant, Dr. Bryant, at my suggestion, cut large openings into four sides of his aluminum balls. This allowed the contracting sclera to dip into these openings in such a way as to make the expulsion of the ball a practical impossibility. Unfortunately, the aluminum proved so unstable as to be useless. But when one of the patients into whose evisceration stump I had put one of these balls returned on account of irritation, caused either by the decomposing metal or by the sharp points which were left by the process, I found it so tightly clasped by the sclera, that it was removed only with the greatest difficulty. Doubtless such a fenestrated shell

made of gold or of some other stable material, (or the wire cage suggested by Landmann[10]) would be well retained; but such good results can be obtained with glass balls by the subconjunctival incision combined with permanent or semipermanent sutures, that I have not thought it worth while to pursue the fenestration principle further. A theoretic objection to any device of this sort is, that unless it fills up perfectly solidly, it leaves a cavity in which liquids can collect and perhaps become decomposed or infected.

Dimitry[11] has proposed a most original innovation in the way of an evisceration implant. After an evisceration, in which he combines the retention of the cornea and an incision entirely scleral with the transcleral neurectomy of Huizinga, he inserts a gold and enamel eye shaped implant. On the front end there is a representation of an iris and pupil. Dimitry hopes that the cornea will remain sufficiently transparent to reveal the details of this picture, and thus avoid the necessity of an artificial eye. A collar button shaped projection passing thru the posterior scleral opening keeps the implant from changing its position within the cavity. This implant is highly ingenious, but from my own experience with the transparency of retained corneae in evisceration, I am afraid it will not appreciably cut down the demand for artificial eyes.

To obtain the best covering for a ball, introduced after an enucleation, the writer has practically nothing to add to the various suggestions and technic described in the excellent article by Frank Allport in the American Encyclopedia. It has, however, occurred to me that an extragood conjunctival flap to cover the deeper sutures could be obtained by excising small triangles at the ends of the horizontal diameter of the conjunctival opening; then, after making a short vertical cut upward from the ends of the main opening, draw the upper conjunctival flap about one-fourth inch or more down below the incision, having first destroyed with trichloracetic acid the epithelium of the surface to be thus covered.

The Use of Foreign Bodies in the Treatment of Glaucoma.

The operation proposed by Mayou[12] and by Zorab,[13] of introducing a loop of silk thread into the anterior chamber and leaving it there, in the hope that it will produce a permanent subconjunctival fistula, probably has some merit. I saw two

successful results obtained by it in Zorab's practice; but as it failed in two of my cases in which everything else had failed, I have not tried it further. In performing it, the main difficulty seems to be that it is hard to let go of the loop of thread when it has once been introduced into the chamber by the forceps. To meet this, Casey Wood[14] has devised a knife with an open eyelet on one side, which drags the thread into the chamber. I have found that a spatula with a notch in the end, as suggested by Mayou, serves very well in pushing the loop into the chamber. Stephansson,[15] recognized the value of the seton operation of Mayou and Zorab, rejected the silk loop, apparently because Zorab has lost one case by panophthalmitis, and has substituted for it a gold wire implant twisted into the form of a T, with a vertical spiral component which is inserted into the anterior chamber. He has also tried, without better results, a T shaped gold tube. Altho the validity of his premise, that silk is more dangerous than gold wire, is doubtful, it seems probable that there might be less chance of filling up the grooves in a gold spiral than of the spaces between silk threads; and the ease with which the wire insert can be moved up and down in case the drainage stops, indicates that Stephansson's device may have a distinct advantage.

Foreign Bodies as Windows for the Eye.

It may have escaped some of the younger members of the society, that many years ago some one, whose name I have lost, attempted to restore sight to eyes with opaque corneae, by putting something like a glass collar button into a hole in the sclera. That this was unsuccessful need hardly be mentioned, but it is worth while to add, that more modern attempts to make windows in opaque corneae, by implanting grafts from the cornea of rabbits or from other humans, has hardly been more successful. In a biologic sense, such grafts are foreign bodies, and the immediate results, which give such joy to the hopeful patient, are practically always vitiated by the opacification of the graft. So strong is my belief in the impossibility of obtaining permanent results from the use of corneal hetero- or homografts (autografts are a different matter), that I hereby offer a prize of fifty dollars to any operator who can show a case in which useful vision has been restored by such grafts for a period exceeding three years. Temporary re-

sults are often obtained in this way, but these are due to the peculiarity of the cornea which enables it to retain a certain amount of transparency even after it has been dead many months. This has been abundantly shown by Salzer,[16] who found that he obtained practically as good results by corneas which had been preserved in formalin, as by grafts immediately transferred from the living animal. My belief in the impossibility of making any such grafts live was obtained from numerous experiments with Thiersch grafts. I found that autothiersch grafts invariably lived, while homografts, even when taken from my own sisters, just as invariably failed to live. Such grafts appear to live and may undoubtedly act as a framework for new formed tissue in the process of healing but when they are watched carefully, a dead pellicle can nearly always be seen to peel off after some days, while with the autografts no such pellicle is shed. A better way to demonstrate the difference in the viability of auto- and homografts, is to use pigmented skin either from whites or Africans. An autograft from a permanently pigmented spot heals and retains its original color, while a homograft apparently heals, but loses its color. The whitest spot on my body is the site upon which I implanted a graft from a coal black negro. The healing apparently was perfect, but the color didn't stick. Another way to demonstrate the lack of viability of such foreign bodies as homografts, is to implant skin grafts into the inside of the lips. The homografts disappear, while the autografts, however small, remain as permanent islands of epidermis in the mucous membrane. When such simple structures as epidermal grafts fail to live when transplanted to another body, it can hardly be expected that corneal tissue would survive a similar test. It is needless to say that egg membrane and similar substances, while they may serve a useful purposes in promoting healing, are never incorporated into the body.*

Foreign Bodies in Lacrimonasal Surgery.

While the value of probes in the treatment of lacrimal strictures can not successfully be questioned, my experience indicates that the attempt to restore the canal by the use of

*These conclusions as to the nonviability of homo- and heterografts do not apply to animals as low as amphibians, especially the immature forms, in which tails and eyes seem to be freely transferable.

stilets, or to substitute hollow tubes for the natural canal, is no good. Ever so often, someone bobs up with a duck bill or some other ornithologic device for carrying off the tears. In my experience, these tubes are stopped up at the ends nearly all the time, and function only as stilets; and this applies even when they have a lumen of two or three millimeters. A permanent stilet will keep the canal open, but it also keeps up watering and some suppuration. Furthermore, as we all know, they are prone to disappear in the depth of the sac, and altho they can generally be fished out without much trouble, occasionally this process of salvage is by no means easy. I have recently had such a case, in which the stilet had been put in twelve years ago, and both it and the patient disappeared within a month or so. In the intervening period, it had caused no especial trouble except that the eye continued to be watery and mattery. It could be felt with the probe in the depth of the sac, but I made no progress in removing it until a radiograph was taken, which showed the short arm to be in an entirely different position from what I had expected. Then, by enlarging the opening into the sac, it was easily removed with the aid of a small strabismus hook. It was found to be perfectly bright and uncorroded, except for the eighth inch which had reached into the nasal cavity. This had a black coating, probably of some sulphur compound.

I still use lacrimal stilets occasionally in the initial stages of a probing cure. Sometimes, with very sensitive patients, the process of cocainization is much facilitated by leaving a stilet in between probings, so that on removing it cocain can be syringed thru without any preliminary probing, and where the further probing has to be entrusted to the family physician, false passages are less likely to be produced if the stilet is put in after each probing, until the stricture has become permanently dilated.

As a curiosity, I may mention the practice of a European colleague, who hopes to cure lacrimal strictures by passing a thread thru the canal and tying the ends together on the cheek, leaving the thread in place for months, sliding it up and down occasionally. If he would have a graduated series of beads strung on the thread, and if submissive enough patients could be found, it might work after the manner of an esophageal dilator.

The Use of Paraffin in the Lids and Orbit.

In the heyday of paraffin injections, I once got a very good result from such an injection, in the smoothing out of a deep sulcus above an upper lid which covered a glass eye. I lost track of the man after a few months, and can not say how long the improvement lasted. If I had to treat such a condition again, I should try a fat implant. Such implants have given me excellent results in cases of the depressed forehead sometimes bequeathed to us by our rhinologic friends; also in the depressed scars of the lower margin of the orbit. The main points in the technic are to make the incision a short distance away from the margin of the depression, and to put in plenty of fat and to unite the cut very carefully. During my early enthusiasm about paraffin injections, I once proposed to a lady to cure her traumatic enophthalmos by this method. Luckily she did not accept. The adhesions would almost certainly have prevented any great improvement in the appearance, and the pressure might have spoiled her sight.

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OPHTHALMIC PLASTIC SURGERY—SUGGESTIONS REGARDING PROCEDURE IN CERTAIN CASES.

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No attempt is made in this paper to cover the whole field of plastic surgery of the lids and orbit. Only such methods of procedure will be considered as have come under the observation of the author in sufficient number to enable him to form an opinion as to the value of a certain procedure in a given case. This does not mean that other procedures may not be equally successful, but that the average results have been more satisfactory by those adopted.

The discussion which follows will be considered under the following headings:

1. Choice of grafts and flaps.
2. Cutting epidermic grafts.
3. Management of grafts and flaps.
4. Correction of certain deformities.

CHOICE OF GRAFTS AND FLAPS.

The choice of grafts and flaps has been governed largely by the fancy of the operator, rather than by consideration of the physiologic function of the part involved. In reference to this point, I quote by permission an opinion recently given by Dr. V. P. Blair:

"In the parts of the face that are most concerned in expression, the brow, the eyelids, the alae of the nose, the lips, and to some extent the chin, the muscles concerned lie almost immediately under the skin or are attached to the skin. In replacing the lost skin in these parts, it should be a free skin graft carrying no subcutaneous tissue, for any appreciable amount of the latter will nullify the action of those muscles. This refers particularly to the eyelids and lips, where the full thickness of the skin has been destroyed, but where at least some of the muscles remain."

Wheeler also has emphasized this same point, by recommending that the skin of the brow or lid be utilized in lid repair.

While there seems to be a general tendency in favor of using whole skin grafts, I am still of the opinion, that in lid repair, epidermic grafts may frequently be employed with good ultimate results.

Cutting.

The cutting of epidermic grafts will be facilitated by smearing the prepared surface and the razor, which must be sharp, with a layer of sterile vaselin. By this means, a large thin graft that will not curl up, and is easily applied to the prepared surface or to the mold, can be readily cut.

The use of vaselin in cutting epidermic grafts is advocated for the following reasons:

1. Very thin grafts that are regular in shape are more readily cut.
2. Grafts do not curl up and are, therefore, more easily handled.
3. The grafts tend to adhere to the mold or prepared surface.
4. The healing process is not disturbed.

Management of the Graft.

Transfer the graft directly from the razor to the surface to be grafted. This can be readily done after putting a few drops of a saline solution on the razor and encouraging the solution to flow under the graft.

If the surface cannot be covered by a single epidermic graft, others may be applied, overlapping the original one 3 or 4 mm. When grafts are to be applied to a mold, they should be adjusted so that there is the least possible danger of disturbing them when placing the mold in position. That is, the line of overlapping should be in the line of force applied to introduce the mold. This suggestion applies especially to operations for cul-de-sac restorations.

After the epidermic grafts are in position, one of several details may be observed. If the graft is large enough to cover the whole surface to be grafted, after the dressings are applied, no sutures are necessary. On the other hand, if there is the

least probability of the graft being pressed away from the edge of the prepared surface, sutures should be inserted in sufficient number to insure perfect contact. If sutures are necessary, the needle should be entered thru the graft first, then thru the edge of the skin. By this procedure, the sutures are more easily introduced and there is less danger of disturbing the graft.

In general, if the conjunctiva is intact, the use of epidermic grafts is to be preferred. On the other hand, if the whole lid is involved in the destructive process, whole skin grafts are to be employed.

Correction of Certain Deformities.

Ectropion. Ectropion may be partial or complete, and either lid may be affected. The surgical management of the senile form of ectropion, due to hypertrophic conjunctivitis, has been reduced to one of two procedures. When the ectropion is of moderate degree, the Ziegler galvanocautry puncture will frequently suffice. When employed, the puncture must pass thru the tarsal plate. If the Ziegler puncture fails to correct the ectropion, it may be repeated, or if eversion is extensive, a blepharoplasty is resorted to after the method of Kuhnt-Szymanowski—a combination of sliding the lid outward together with the removal of a triangular piece of cartilage and conjunctiva. In removing the triangular piece of skin, the base of the triangle should run up and out from the external canthus, at an angle of 45°, and not horizontally, as is so often illustrated. Excision of a portion of the skin, or of the tarsus and conjunctiva alone, has not proved, in my hands, so successful as the combination of the two procedures after the method of Kuhnt-Szymanowski.

Scar tissue contraction ectropion may vary in degree from a slight linear scar to a complete evulsion of both cul-de-sacs. When the scar is small and linear and within the lid, it may be resected and the lid margin restored by the socalled V Y operation. This procedure, however, is effective only when a small portion of the lid is everted. My impression is that I have had quite as many unsatisfactory as satisfactory results following operations after this procedure. As stated in Spaeth, "It will assist very much in the placing of the sutures if the two linear incisions are made continuous with the base of the triangle for a distance of 3 or 4 mm., bent downward

slightly, and then undermined, it being a modification first mentioned by Dieffenbach."

When the ectropion is extensive and the conjunctiva is intact, the most satisfactory results have been obtained by the employment of epidermic grafts after the following procedure. An incision is made along the lid margin (wherever it may be located) immediately outside the hair line. The lid is dissected free, care being taken to keep the lid thin. The dissection should be so free that the lid will rest in its proper position without traction. If the correction is to be made in the lower lid, it should be turned over the upper lid and held in this position of overcorrection by sutures passed thru the lid margin and attached to adhesive tape on the forehead. After the bleeding has been controlled, the whole surface is covered with an epidermic graft. The grafts should overlap the skin edges 3 or 4 mm. If for any reason the graft does not "seat" perfectly and remain in position, sutures may be introduced. Otherwise, the dressing may be applied. If both lids are everted, a second operation following the same procedure is necessary. Occasionally, in extreme cases, a third operation is necessary to restore the canthus. Even so, each operation requires but a week or ten days, and later, if necessary, the canthus can be restored. By this procedure, the inconvenience to the patient, incident to having the lids closed for a period of months, is avoided.

The form of dressing to be applied will vary with the character of the surface to be grafted. If the surface is small and smooth, the graft will take if no dressings are applied. Experience has proved, however, that the immediate results are better if pressure is used. If the surface is large and not crater like, the graft may be covered with strips of rubber, about 10 mm. wide, laid on overlapping, or if a little more pressure is needed, a disc of dental material, or of wax and paraffin in equal parts, may be molded over the graft and surrounding parts.

If the surface is uneven or crater like, or if the surrounding skin shows a tendency to overlap, an impression of the prepared surface may be taken by pressing into it soft dental material or a combination of wax and paraffin. This procedure insures perfect contact of the graft and the area to be grafted, as the mold, pressed into the wound, smooths out the soft tissues and reaches the uneven surfaces, thus advantageously

enlarging the area to be grafted. After the mold sets, it is removed and the surface of the mold bearing the impression of the area to be grafted is covered with an epidermic graft slightly overlapping the area, and the mold replaced. Gauze saturated with sterile vaselin is then overlaid, all held in place by a bandage.

On the eighth day, dressings are removed and the parts cleansed with a warm boric acid solution. The stay sutures are taken out and the dressing reapplied for three days, at the end of which time all dressings are removed and the graft smeared with sterile vaselin or other ointment.

The question as to the amount of pressure exerted by the bandage has not been seriously considered. The manner in which the bandage is applied may be described as "snug," which is defined as compact, convenient and comfortable.

If the whole lid is involved in scar tissue, or if it is partially or wholly lost, the pedicled flap may be required to correct the deformity. The flap may be taken from the adjacent skin in any direction, but in general, the pedicled flap for the correction of the lower lid should be taken from above, and that for the upper lid from below. By this procedure, the pull incident to the contraction tends to hold the lid in contact with the globe, rather than to draw it away, with a resultant ectropion. While a graft will take if it contains subcutaneous tissue, in order to secure the best physiologic result, the flap should be made as thin as possible. Moreover, if the flap contains only the whole skin, the tendency to pucker at the base is diminished. This tendency can be further lessened by freely undermining the skin around the base.

After the flap is cut, allowing about one-third for contraction, it should be adjusted so that it will remain in its proper position without undue traction. No greater mistake can be made than to force the position of a graft by the use of sutures. Time must not be an element in the operation.

Many operations have been suggested for the correction of lid deformities, and in special cases great ingenuity has been shown in the selection of flaps. Valuable suggestions are found in Spaeth's book on Ophthalmic Plastic Surgery, and in articles by Wheeler.

SYMBLEPHARON: Because of the seriousness of this condition, every effort should be made at the time of the accident (almost always traumatic or chemical in origin) to prevent the forma-

tion of adhesions. One fruitful means of prevention would be to have all shop cases treated by oculists instead of by shop nurses or others unskilled in the management of this class of cases.

The surgical management of symblepharon is often very unsatisfactory, in spite of our best efforts. The use of whole mucous grafts is frequently advocated, but they are difficult to manage. Mucous membrane grafts taken from animals are not satisfactory. I have attempted to graft the conjunctiva from rabbits to humans with no success. The conjunctiva from humans, however, can be utilized for conjunctival repair. As only small mucous membrane grafts are procurable, it is sometimes necessary to use the superficial layers of the skin. When this procedure is followed, there may be a troublesome exfoliation, but the inconvenience to the patient is nothing compared to the symptoms incident to the presence of a symblepharon. Epithelial grafts may be used on a mold, and, so far as I know, Dr. Wilder was the first in this country to suggest the method of their application.

If the symblepharon is extensive, a disc thru which a hole has been cut to protect the cornea can be covered with an epidermic graft and inserted over the globe, after the lids have been dissected free. I have used a disc made from a mixture of wax and paraffin in equal parts. These ingredients are melted and allowed to cool, when a mold can be made of proper size. It never becomes very hard, and consequently is less prone to injure the cornea than dental material or other hard substances.

Cul-de-sac Restorations: The essentials in this operation may be described as follows: a thin lid, extensive dissection beyond the orbital margins at the angles, to the orbital margin below and nearly to the orbital margin above, removal of all scar tissue, and a well fitting mold. Whether or not the periosteum at the angles should be incised, I am not certain.

The lid should be incised inside the hair line, care being taken to keep it thin and of the same thickness thruout the entire length. The dissection at the canthi should go beyond the orbital margin. The position of the inferior punctum must be given special attention and care exercised not to injure the canaliculus. The inner portion of the lid may have the appearance of a slight undercorrection after the mold is in place. In special cases, I have found, when the palpebral

fissure is small or a large mold required, that the insertion of the mold is greatly facilitated by incising the outer canthus. Usually no repair of the outer canthus is necessary. It takes care of itself. The importance of the removal of all scar tissue cannot be overstated; it is essential for success.

After the dissection of the lid is completed, a mold is made of dental material. A convenient way of estimating the approximate size of the mold is by using a fixation forceps as a pair of calipers. Separate the forceps by pressing the finger between the blades. Measure the maximum distance from the upper to the lower orbital margin. Mark this on the mold by drawing the forceps over its surface. Repeat the same procedure for the horizontal measurement, marking the mold at right angles to the vertical measurement. This will result in a rectangular figure that will enable one to quickly cut a mold of approximate size. The mold required is usually of smaller size than one would expect, and I am sure you all have had the experience of beginning with one much too large.

It is not necessary to make the mold more than 3 or 4 mm. thick. The edges should be tapered off and a thin graft carefully applied. If only one sac is being restored, an incision may be made in the graft about 5 or 6 mm. from the lid margin that is being restored, and the graft turned out over the lid margin. By this means, granulation of the small uncovered areas on the lid margin may be avoided.

The mold should be so fitted that the lid closes easily and completely over it. The lids are held together by the use of a mattress suture introduced thru the skin of the lids, 3 or 4 mm. from the lid margin.

The dressings are left in place eight days. If at the end of this time there are no complications, the skin sutures are removed, the discharge washed away and the bandage reapplied for two days longer. Then the dressings are removed and the orbit is washed out daily. The exfoliating epithelium can be removed by the use of alcohol on a cotton pledget. The amount of discharge present at the time of the first operation seems to depend on the amount of normal mucous membrane left in the orbit. The more radical the operation the less the discharge.

I have had no experience with the use of pedicled flaps for the cul-de-sac restorations. In my hands, better results are ob-

tained by the use of epidermic than by whole skin grafts after the method suggested by Weeks.

I have also had no experience with tubulated grafts, and not enough with bone grafts to justify any suggestion as to their use.

Glass Ball Implantations: For several years I have followed the suggestion of Lancaster to omit the suturing of the muscles over the glass ball. I am of the opinion that the average results are fully as satisfactory, if not better, than they were before this procedure was followed.

THE USE OF FOREIGN BODIES IN EAR, NOSE AND THROAT SURGERY.

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Inasmuch as I am to discuss one of the phases of plastic surgery, I deem it of sufficient interest to my audience to give a very brief history of this subject from the time of Celsus down to the present century. In reviewing the recent literature on plastics, one is astonished at the large number of authors who have devoted entire papers to the history of this subject alone. In 1909, Otto Hildebrand[1] published a monograph entitled "Die Entwickelung der plastische Chirurgie," and the following statements are quoted largely from his paper.

Celsus was the first and only one of the ancients to mention plastic surgery. He wrote entirely on plastics of the nose, lips and ears, and did not go into general surgery. None of the other noted Greek, Roman or Arabian physicians seemed to know anything of this subject, for nowhere is mention made of this topic. So far as Europe is concerned, this subject lay dormant until the fifteenth century, when two Sicilians, Branca or Brancas, father and son, revived plastic surgery. They did a great deal of work secretly. It is known, however, that the father used flaps from regional parts, while Branca, Jr., who did the major portion of this work, obtained the flaps from the arms of his patients. This latter procedure has since been known as the "Italian Method," and is still used extensively by modern surgeons. They were followed by two Calibreans, Bojani Brothers, who also used the arm flaps. During the next two centuries, nothing was published concerning the work, altho it is known that plastic surgery was performed extensively in Southern Italy. Late in the fifteenth century, Taglizcozzi, professor of anatomy and medicine in Bologna, published a lengthy treatise on plastic surgery, which gave inspiration to contemporary surgeons, and he is now considered the real founder of plastic surgery. Drawing nearer

our own time, Graefe was the first of our modern surgeons to devote his time and energy to plastic work, and he highly recommended the Italian Method, while Diffenbach, by his life's work and interest, gave the greatest impetus to modern plastics. We find that plastic surgery was being performed for many centuries in India, altho it was unrecorded in medical literature. It is known that they employed regional flaps. Hence this procedure is known in our time as the "Indian Method." It is of interest to know that India anteceded other countries in the use of plastics, because one of its commonest forms of punishment, for even trivial offences, was the slashing off of the culprit's nose. The criminals, anxious to assume their place in society, sought medical aid in the restoration of their features, making facial plastics a necessity. Judging from this short history, we are led to believe that very little was published concerning plastic surgery before the middle of the past century. Yet Edward Zeis,[2] in his book on plastic surgery, published in 1863, recorded and abstracted two thousand and eight articles written prior to that time, proving that the modern medico is not unique in presenting prolific records of the work he is doing.

Arriving now at my portion of this symposium, namely, "The Use of Foreign Bodies in Ear, Nose and Throat Surgery," I find in the literature mention of many types of foreign material, such as gold, silver, vulcanite, plaster of paris, sealing wax and decalcified bone, and in practically every case their use was confined to cranioplasty, with but little mention of their employment in plastic surgery about the face. Until the present century, all plastic surgery about the face had been done exclusively with the aid of skin flaps, and without the support of any underlying framework. Since then, three types of foreign materials have been extensively used by modern surgeons, namely, paraffin, celluloid and ivory, which I shall discuss in this paper.

Vaselin and Paraffin. R. Gersuny,[3] in Die Zeitschrift für Ohrenheilkunde, in 1900, published a paper entitled "A New Subcutaneous Prothesis", describing the use of vaselin. He tells of a patient whose testi he was obliged to remove because of tuberculosis. Returning to him a year later, seeking the Doctor's help in avoiding ridicule to which his call to army service would subject him, Gersuny injected vaselin into the scrotum with a perfect cosmetic result. Later, he used

vaselin in cases of cleft palate, by injecting it in the uvula and posterior pharyngeal wall. In cases of saddle nose, he used it successfully and highly recommended this method. Following Gersuny's article, paraffin was substituted for vaselin, and was employed very extensively until the evil aftereffects became known, when it was discontinued by all ethical surgeons. We have all seen some of the bad results from its use, and I herewith quote from a resumé of its dangers as cited by Dr. Joseph Beck[4] in his contribution on plastic surgery to Loeb's "Operative Surgery of the Nose, Throat and Ear:"

1. Toxic absorption.
2. Marked inflammatory reaction.
3. Loss of tissue due to infection and abscess formation.
4. Pressure necrosis caused by hyperinjection.
5. Sloughing of tissue as a result of the heat of the ma-
6. Sloughing due to injections into very dense or inelastic structures, or where scare tissue is firmly attached to underlying adjacent parts.
7. Subinjection or too small an amount of paraffin, with an insufficient correction of the deformity.
8. Hyperinjection with overcorrection of the deformity.
9. Air embolism.
10. Paraffin embolism.
11. Primary diffusion, or extension of paraffin when first introduced, into adjacent nasal structures.
12. Interference with muscular action of nose.
13. Escape of paraffin after withdrawal of needle.
14. Solidification of paraffin in the needle.
15. Absorption or disintegration of the paraffin.
16. Difficulty in procuring paraffin at proper melting point.
17. Hypersensitiveness of skin over injected area.
18. Redness of skin over the injected area.
19. Secondary diffusion of the injected mass.
20. Hyperplasia of the connective tissue following the organization of the injected matter.
21. A yellow appearance and thick lining of the skin after organization of the injected matter.
22. Breaking down of the tissue and a resulting abscess, due to pressure of the injected mass upon the adjacent tissues after the injection has become organized.

There is no question that all these difficulties and dangers are authentic, and every one may occur in the practice of a plastic surgeon where paraffin is used to a great degree. Another danger not specifically enumerated is the formation of a paraffinoma, which we class as malignant, but not in the sense of a carcinoma or sarcoma. There is a gradual infiltration of connective tissue into adjacent structures, as for instance, when injected subcutaneously for a depressed saddle nose; the paraffinoma attacks the orbital cavity, and may cause serious injury to these structures. As examples I cite two cases. The first, Miss N., a young lady, twenty years old, consulted an ethical rhinologist regarding the appearance of her nose. She was advised to refrain from any interference, as the nose was perfect. Notwithstanding this, she consulted an advertising charlatan, who immediately injected paraffin subcutaneously. For a short time she was apparently pleased with the result, but within a year a reddening and irregular surface at the site of injection was noticeable. This irregular red mass gradually spread over her forehead into her cheeks and into the orbital cavity, accompanied by severe pains over the entire region. With this picture, she came to us for relief, both for pain as well as for cosmetic reasons. The overlying skin was infiltrated and so firmly connected with the paraffin, that it became necessary to remove it with the tumor. Large masses of paraffin were removed from the whole region, with the result, that while the pain was relieved, the appearance of the area was unsightly and much depressed. Subsequently, a flap from the arm was obtained which covered the defect, but it was white and has ever remained so, while the adjacent skin is red. Two attempts to elevate the depressed graft, one by implantation of bone, the other of ivory, resulted in failure, as the tension was too great, due to the fact that we were dealing with too much scar tissue. The other case is one of multiple growths due to injections of paraffin in various parts of the face and neck. This lady, a graduate nurse, not liking the wrinkles in her face and neck, decided to do her own injecting, result of which I shall show later in a lantern slide.

Years ago, my associate, Dr. J. C. Beck,[5] following the recommendation of von Stein, a Russian rhinologist, injected paraffin into the inferior turbinate bodies in cases of atrophic rhinitis. He reported his results in the Annals of Otology in 1907. In the seven injected cases, he temporarily obtained

good results, but in a very short time, the paraffin sloughed out, leaving the condition of the nose the same as prior to the injection. Some time later, we conceived the idea, that if we could place the paraffin under the skin or mucous membrane without injecting it, most of the dangers could be averted. I reported a series of cases in which I obtained excellent results in atrophic rhinitis, by using paraffin inlays under the mucoperichrondrium of the septum. While we had no complications from this procedure, the end results were unfavorable, as nearly all of these inlays eventually sloughed out.

Celluloid, the second of these heterogenous substances, has not been extensively used. Oscar Föderl,[6] in the Wiener medicinische Wochenschrift, in 1903, was the first to suggest the use of this substance in plastic surgery, and he recommended it only to cover cranial defects. H. Koshier,[7] in the Wiener medicinishe Wochenschrift, in 1908, advised its use in plastic surgery about the face. He used it as a framework in reconstructing a new nose. He shaped his celluloid first, and then buried it under the skin of the forehead, and after it had become attached, he made his flap from the forehead including the celluloid inlay, and turned it down to construct the nose. He reported two cases, one in which the celluloid sloughed out and the other in which the celluloid remained in place until the patient died, one and one-half years later, from an intercurrent disease. In the Journal of the American Medical Association of April 16, 1918, Dr. Gordon New[8] reported his experience with this material. He had the celluloid perforated, trusting that the connective tissue would grow thru these openings and hold them in place. In four of the six dogs on which he experimented, the inlays sloughed out, while in the other two, they remained in situ. When removed, it was found that the connective tissue had grown thru the perforations. He then tried it in five cases of saddle nose, with good results. Desirous of knowing the ultimate results of these implants and finding no reference to same, I wrote to Dr. New and was informed that he had discontinued the use of celluloid in plastic surgery, as all of those reported cases had sloughed out within a period of three years. In reviewing the literature, I found no further reports on celluloid, so that we must conclude that it has been an utter failure in plastic surgery.

Ivory, the third substance, has been used by European sur-

geons for a number of years but it is only recently that it has been used in America as framework in plastic surgery about the face. Jacques Joseph[9] in the Deutsche med. Wochenschrift, in 1919, states that inasmuch as bone transplants often become absorbed, he has substituted ivory implants in the treatment of saddle noses, but at this time he was not prepared to give any data. He has been using them almost exclusively ever since (1919), and Maliniak,[10] in the Archives of Otolaryngology, 1925, states that while with Joseph for a year and half, he had occasion to examine some of the earliest cases implanted, and found that they were still in place. In fact, he states, he never saw a single case slough out.

Bone and cartilage have been employed almost universally, the bone being obtained from either the tibia or rib, and the cartilage from the costal cartilage, the ear or occasionally from the nasal septum. From our personal experience, we consider ivory preferable to either bone or cartilage. Ivory, as obtained from the tusks of elephants, is similar in its construction to bone. If anyone is particularly interested in the chemical and microscopic examination of ivory, I refer him to Maliniak's paper, in which he gives an exhaustive study of the chemical and microscopic comparison of bone and ivory. He also draws attention to a "vegetable ivory," which is obtained from the kernel of the nuts of the Lagua Palm tree (Phytelephas macrocarpa). He believes this vegetable ivory superior to the natural ivory, but we have had no experience with the former and, therefore, cannot give a comparative study. We found ivory preferable to bone for the following reasons:

(1) Ease of obtaining implant. If bone is employed, it must be obtained from the tibia or rib, causing the patient to undergo a double operation, one for obtaining the implant, and the second, the plastic operation. When the implant is obtained from the tibia, there is first a risk of infection with long continued suppuration and the danger of an osteomyelitis; second, continued pain in the region of the wound; and third, spontaneous fractures. Cases of spontaneous fractures have been reported, even if only a small portion of the crest has been removed. If the implant is taken from the costal cartilage or rib, then there is risk of infection of the rib wound, with a resultant osteomyelitis of the rib or sternum, or empyema, with a prolonged convalescence. If the cartilage is obtained from the ear, there is a risk of infection resulting

in a chondritis or perichondritis, thereby causing a deformity of the ear. If the cartilage is taken from the septum and infection occurs, a septal abscess with all its evil aftereffects, may follow.

(2) Exact shaping of implant: (a) in shaping or bending implant of bone, there is the risk of breaking and spoiling its usefulness; (b) the difficulty in obtaining a piece of bone or cartilage of the desired size, as frequently the implant is too small and an additional piece is necessary. Our experience shows that a single implant gives better results than where numerous pieces are used. When using ivory, we can measure the size that is needed with a great deal of accuracy, tho should it prove to be too large at the time of operation, it may then and there be reduced to the desired size. We always have many pieces sterile and ready for use, and it is an easy matter to select the required one. The edges of the bone transplants are often rough and sharp, sometimes causing them to penetrate the overlying skin. This complication does not occur with the use of ivory, as its edges can be made as smooth and round as desired.

(3) In sterilization, ivory, unlike bone or cartilage, can be boiled and put thru the strongest disinfectants without affecting its usefulness.

(4) Absorption of the implant. There is little or no chance of the ivory implants being absorbed for now, more than seven years since these transplants have been used, there is no record of even the earliest cases disappearing. On the other hand, there has been and still is much discussion as to the absorbability of bone and cartilage. Most authorities agree, that if bone with its periosteum or cartilage with its perichondrium be used as implants and brought into direct contact with living bone, there is little danger of its being absorbed. In using these tissues for plastic work about the face, it is necessary often to bury them in soft structures, and it is then that they are likely to become absorbed or degenerate into fibrous tissue. Again, it has been observed that occasionally they hypertrophy, especially the cartilage, thereby causing a deformity. We had such a condition in a physician who had had costal cartilage implanted for a saddle nose. Within two years, there was a large hump due to the hypertrophy of the cartilage, and a second operation was necessary for the reduction of the hump.

The disadvantages of ivory are twofold. First, it is a foreign material, and does not grow or live like bone or cartilage and become an integral part of the body. It has been shown that it eventually becomes encapsulated by a firm fibrous capsule, just as other foreign bodies, such as bullets, pieces of steel, etc., do. Owing to the fact that it does not become an integral part of the body, there is a greater likelihood of its becoming displaced, which condition we have occasionally observed. At present, we are perforating the ivory implants, with the hope that the connective tissue will grow thru the small openings, holding them firmly in proper position. Second, the danger of the implant sloughing out must be considered. It is now over three years since we have been using ivory implants frequently, and thus far only a few have sloughed out and those particularly in cases where primary infection occurred. As previously stated, reports coming to us from European clinics corroborate our finding, with the additional fact that they have observed these cases for a period of over eight years.

To describe all of the various plastic operations in which ivory is used, is not within the province of this paper. Suffice to say, that wherever and whenever bone or cartilage has been or can be used, ivory makes an excellent substitute, with all the advantages I have enumerated. I shall present a number of slides demonstrating the ivory in situ in various plastic operations. I wish to call your attention particularly to the slides depicting ivory implants in cases of atrophic rhinitis, demonstrating their adaptability in the treatment of this disease. Three years ago I presented a paper before the Triological Society entitled "Intraseptal Implantation in Atrophic Rhinitis,"[11] in which I described my method of implanting bone and cartilage under the mucoperichondrium of the septum in the treatment of this condition. I reported excellent results by this treatment, but explained the difficulty in obtaining the implants. During the past year and a half, I[12] have been using ivory implants instead of bone, and in May, 1926, presented this method and its splendid results to the Chicago Laryngological and Otological Society. This will appear shortly in the Annals of Otology.

Conclusions.

(1) Foreign materials, such as gold, silver, vulcanite,

plaster of paris, decalcified bone, paraffin and celluloid have been tried in plastic surgery about the face, and have all been discarded by ethical surgeons. (2) Ivory, in our experience thus far, is an ideal material to use as a framework in plastic surgery, and is superior to either bone or cartilage. It is, however, still too early to make definite assertions as to the permanency of results from the use of this material in plastic surgery, tho we feel much encouraged by the reports of our German colleagues, which lead us to believe that we need not fear the absorption or loss of these implants.

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DISCUSSION OF SYMPOSIUM ON PLASTIC SURGERY.

DR. JOHN M. WHEELER, New York City: In cases of depression, such as Dr. Gifford spoke of following external operations on the frontal sinus, let me suggest the use of deep fascia instead of fat, which, as he says, is invaded with connective tissue. For depressions in bone, of course we are all familiar with the use of grafts of bone, cartilage and other material, but in my opinion and experience there is no tissue which is as generally satisfactory for filling as fascia lata. This tissue remains as fascia. In one case in which I got an overcorrection after transferring fascia lata for a depression from bony loss, below the orbit, I opened the original wound and exposed the fascia to reduce the amount about a year after I had transferred it. It was possible at that time to pick up the fascia and remove it almost as tho it had been put in the day before. It looked almost as it did when it was put in.

I am nearly in accord with the views which Dr. Parker has expressed, and I was quite interested in hearing him read a qnotation from Dr. Blair, expressing the belief, that in plastic surgery such as we are interested in, the use of detached grafts is better than the use of pedunculated flaps. This means a change of front for Dr. Blair, and I think it is important. I wonder if the time is not here when, as American

ophthalmic surgeons, we can take some such forward position as this—that when it is possible to secure a good bed for the reception of the graft, epidermis or true skin without pedicle can be transferred to such a bed with the assurance that the graft will take and will fulfill its function. If we take such a position, I think it will be important, and will be an advanced one.

There are certain conditions in which it is necessary to use pedunculated grafts for ocular plastic surgery. For instance, cases such as that Dr. Parker showed, where the whole upper eyelid was gone and no membrane left of any consequence. No suitable bed could be obtained—the eye does not furnish a suitable bed. In such cases, the pedunculated graft should be used. In case of coloboma of the lid, the same thing is obvious, and I have held that in such cases a graft should be used from the region of the temple, making the dissection as far back as necessary.

There is one point which Dr. Parker made with which I shall disagree, and that is that he advocates epidermis instead of true skin in a case of ordinary ectropion. In every case where it is possible to engraft for ectropion, I should use the true skin of the eyelid. There are at least two good reasons for this: In the first place, it is possible to correct the condition entirely by the use of this skin, and then the result is more beautiful than can be obstained by epidermis, which never would entirely match. Dr. Parker this morning showed a picture of a case which he had operated two years before, in which it is perfectly possible to see the outline of the epidermic graft. So in all cases where it is possible, I should use lid skin. I should, however, use epidermic graft where it is necessary to use tissue away from the lid. Dr. Parker has shown cases in which a very bad facial burn exists along with lid burn, and in such cases the epidermis matches as well as true skin.

I am glad Dr. Parker spoke of the use of vaselin in making grafts. If I am not mistaken, Dr. Parker was the first to suggest it several years ago.

I did not exactly like the impression that Dr. Parker gave in reading the definition of "snug." It is too complacent and easy going. I think I disagree with him, if I understood him rightly—at least the wrong idea is imparted. In grafting about the eye, firm pressure, secure pressure, is very important for several reasons. In the first place, in using detached grafts we are dependent almost entirely on the underlying tissues for vitalization. If a graft is not securely in place on its raw surface and thru its whole extent, it is likely to be a partial failure. For this reason, the firmness of the pressure is important. Furthermore, the control of the hemorrhage is impossible. And forcing the inevitable secretion into the dressing from under the covering of the graft is worth while. But the thing that is most important about it is this—if the graft is not held firmly in position against its bed, granulation tissue always underlies the graft. That is the only way that Nature has of furnishing union, by throwing out granulation tissue. The amount of granulation tissue controls the amount of contraction and apparent thickening of the graft. Dr. Parker showed a case which he had operated twenty years ago, a case that he was not proud of, and he showed it for the reason

that it was large and thick. That was not entirely because the graft was thick to start with. I was not at the operation, but I am confident that Dr. Parker did not put on firm enough pressure, or he would not have had a subsequent contraction and thickening.

As for symblepharon, I agree with Dr. Parker that ordinarily the results are not absolutely satisfactory either to the patient or the surgeon; and if one operates for symblepharon, I think he must be prepared for a partially unsatisfactory result, and the patient almost must be prepared for it.

Dr. Samuel Salinger, Chicago: The use of ivory and other foreign substances in nasal reconstruction had its origin in Continental Europe, and to this day is preferred to the autogenous cartilage or bone transplant. In this country, in the main, cartilage and bone have been more extensively employed, and judging by the reports emanating from various sources, have proven eminently satisfactory. My own experience has been principally with costal cartilage. Having employed it a considerable number of times for the correction of saddle nose deformities, and having seen these patients as late as three years following the operation with no appreciable lessening in the size of the transplant, I have concluded that the procedure cannot be improved upon.

Foreign authors disparage the use of cartilage in the assumption that the transplant is sooner or later absorbed. They evidently believe, that possessing no vascular system, the cartilage cannot receive nutrition from its new bed, especially when it has been previously deprived of its perichondrium, as is our general practice. In connection with this point, I wish to point out the histologic investigations of Pfoff and Linberg, who were able to demonstrate small blood vessels within the substance of the cartilage; also the experiments of Mangold Nussbaum and Lexer and Irwin in the successful incorporation of transplanted cartilage. Koenig and Lexer have demonstrated cases in which the transplants were observed as late as fifteen years after operation, apparently unchanged.

In my own experience, I have had one opportunity of examining histologically a cartilage transplant into a saddle nose in a man who died of an intercurrent ailment some four months after the operation. The slides show the cartilage well imbedded in a fibrous envelope, its cells viable, and every indication of a healthy incorporation of the transplant into its new surroundings. (See Figs. 1, 2 and 3.)

Comparing it to ivory, one can see an immense advantage in having a normal, healthy, flexible tissue, such as properly belongs to that location, rather than a dense, unyielding, foreign body which will always be nothing more than a foreign body. The only disadvantage in the use of cartilage is in the extra operative procedure entailed by its removal from the rib, and the incidental scar and discomfort to the patient. We cannot deny that ivory can be accurately shaped to a plaster model of the patient and be sterilized and ready before the operation, thus saving a great deal of time and simplifying the operative procedure. We have used it several times in the past few months and can testify to that. But whether these advantages should outweigh those we have mentioned in

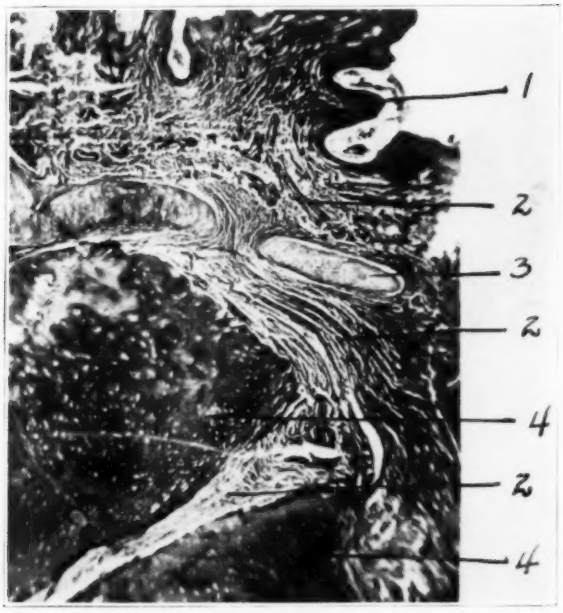


Fig. 1. 1. Nasal mucosa. 2. Fibrous stroma. 3. Lateral nasal cartilage 4. Implanted costal cartilage.

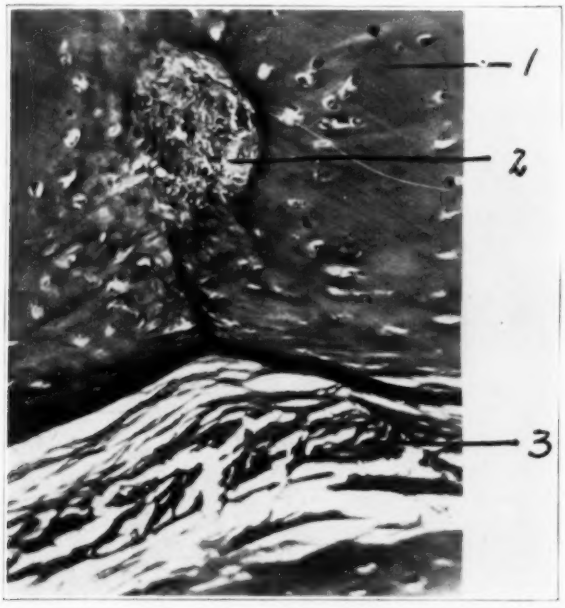


Fig. 2. 1. Implanted costal cartilage showing viable cells. 2. Probably a mass of newly forming fibrous tissue filling defect in cartilage. 3. Fibrous stroma.

behalf of cartilage is something that only the cumulative experience of the next few years can determine.

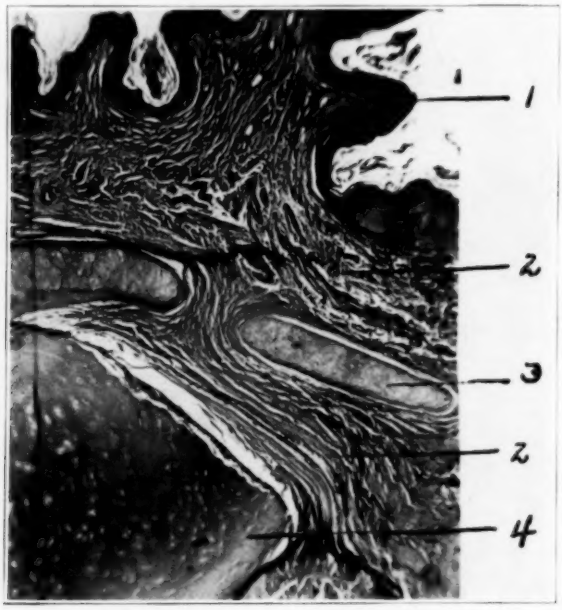


Fig. 3. 1. Nasal mucosa. 2. Fibrous stroma. 3. Lateral nasal cartilage. 4. Implanted costal cartilage.

Dr. William H. Wilder, Chicago: I want to express my appreciation of these interesting and valuable papers on subjects that are of such importance. I think it is well to remember, that the ophthalmic surgeon is favored in his plastic work by having a field that is abundantly vascular, which accounts for many of the favorable results obtained; were it not for the abundant vascularity, our failures would probably be more frequent than they are.

I agree with Dr. Gifford in many of the points he took up in his paper, and particularly in that concerning the use of epidermic graft placed upon the eyeball. They grow beautifully but are not natural there, and I think one who attempts this method will frequently be disappointed, as I have been. Not only do you have to massage or manicure them, so to speak, every week or so, but sometimes you have to shave them, as I have found to my sorrow when I unfortunately included a few hair bulbs in the graft. I prefer to use conjunctiva if it can possibly be obtained. It is difficult to hold in place, but with the exercise of patience and ingenuity, it can frequently be accomplished.

Concerning the subject of Dr. Parker's paper, I should like to emphasize, for the benefit of those who have not had large experience in plastic surgery, some of the points that make for the success of such measures. The first is that the careful removal by dissection of all scar tissue is

very essential to success in cases of ectropion or entropion, and particularly in extreme cases of symblepharon.

Then as to the matter of the graft. In cases of ectropion, my experience has not been as successful with epidermic graft as with one of the whole skin, the socalled Wolfe graft. In that respect my views coincide with those of Dr. Wheeler. But even with the whole skin graft, it is essential that it be large enough, at least one and one-half times the size of the defect to be covered, for it will certainly shrink. Another extremely important point is to spread the graft carefully on some sterile surface and with the help of scissors and knife to dissect away carefully all of the fat and subcutaneous connective tissue, leaving the thinnest possible whole skin graft. Another way to accomplish this is to cut a modified Thiersch graft. Instead of cutting merely the epidermis with the razor, we may go deeper and cut off the dermis itself and so get a thin Wolfe graft large enough in size to cover the defect. It is because of lack of care in these particulars that many of the socalled Wolfe grafts are failures. Again, it is important to have close apposition with a certain amount of pressure, and this can be accomplished by carefully molding a piece of softened dental compound over the grafted area.

As it is so important to the success of such operations to take away scar tissue that has already formed, it seems to me if we can accomplish anything in the way of prevention of the formation of this scar tissue, it will be valuable. This can be done in certain cases of burns of the conjunctiva, as from molten metal or chemicals, which almost inevitably result in symblepharon. After the dead tissue has sloughed away and the defect is covered with healthy granulation tissue, we may attempt to cover the granulated surface with an epidermic graft, just as if it were a burn on the chest or foot or face. The general surgeons have found that it is best, in granulating surfaces from wounds of this kind, to wait for a time, possibly for a week or two, until the shock to the tissues from the acid or the burn has disappeared. On such a granulating area in the eye, we may implant a very thin epidermic graft by folding the graft over a plate of pure lead or pure tin, fashioned to fit the conjunctival sac and coated with hard paraffin to protect the cornea, which may have remained clear and healthy.

I have found the metal plate coated in this way with paraffin superior to dental compound and less likely to injure the cornea. The paraffin used for the purpose should be of the highest possible melting point, so as not to soften too easily from the heat of the body.

Dr. S. Hanford McKee, Montreal, Canada: I come forward simply to emphasize one or two points in Dr. Parker's excellent paper, where he dealt with cul-de-sac restoration. In regard to pressure, I think a firm pressure is most essential in these cases. The restoration of the cul-de-sac has been a difficult matter with us all. The operation of inlay is comparatively easy, but the most important part of the operation is the aftertreatment and the following up of the case for a considerable period. But firm pressure is necessary. My custom for some time has been to do away with the suture and bring the upper and lower lid together. We find, if the compound is large enough to give the necessary pressure

which we feel we should have, it is not necessary then to put in a suture.

Another point which I would like to make is, that any time we have cut the outer canthus, we have always regretted it. I know both Gillies and Waldron have been quite enthusiastic about enlarging the operating field when necessary, but our experience in cutting the outer canthus has been unsatisfactory.

The most important part in restoring the cul-de-sac by the epidermic method is, that after the compound has been removed—from the sixth to the tenth day—and the sac generally cleaned up, it is very important at that time to keep the tissue on the stretch for some weeks. My practice is, that when the compound is taken out—of course it is quite large—we then pare it a little so as to make it enough smaller to go in and out easily. Then we put this compound in the cul-de-sac and leave it there for some weeks. I think that is an important point and I want to enlarge upon it, because I am very glad to admit that in the early part of my work I had some cases that were distinct failures, cases that when the compound was removed looked as beautiful as these cases do, but after some weeks the tissues so contracted that the sac disappeared.

Dr. Ray K. Daily, Houston, Tex.: I would like to suggest a simplified method of fashioning the ivory graft. We found the instruments used by Joseph in Berlin, as illustrated by Dr. Pollock, a very laborious and tedious procedure. Ivory is very hard, and filing it by hand is work of hours. We make a cast of the graft desired with dental compound, which is easily molded and hardens quickly; this cast is taken to the dental laboratory and an exact replica is made within a few minutes from a piece of ivory.

We have been using ivory for three years, and any one trying it is bound to be convinced of the ease and simplicity of its use as compared with rib transplantation. Some of our work was done in the office and the patients treated as ambulatory cases, with good results.

Experience may show that it will not be tolerated by the tissues, as suggested by Dr. Beck. However, one sees it used very extensively in Joseph's clinic, and he says that he has used it for years, without untoward results.

Dr. A. H. Andrews, Chicago, Illinois: I desire to mention only two points. In the first place, in the correction of saddle nose, I have had very excellent results from taking cartilage from the septum and using it to rebuild the nose. It can be shaped as we want, and it makes but one operation. You get away from several of the disadvantages of attempting to take cartilage from the rib.

The other point is regarding Dr. Gifford's paper on foreign bodies. We have a great many patients that have a deep depression with the best artificial eyes we are able to buy on the market. I want to call attention to the fact, that special eye makers, who make artificial eyes for individual patients, are able to overcome many of these defects which are left from lack of volume in the orbits. I recently had a case where I was attempting to reconstruct the cul-de-sac. I called in an artificial eye maker, and he put a projection in the upper margin of the

eye which pushed the eye out and made it look very much better than any eye I could buy on the market.

DR. HAROLD GIFFORD, Omaha (closing): I want to come to the rescue of the Thiersch graft on the eyeball. I think the Thiersch graft on the eyeball has a very decided place in the treatment of recurrent pterygium. A woman who has had one of these things come back several times and has a red eye there, does not mind a bit wiping that flap off once a week—she would wipe it off twenty times a day if it would make her look better. You can get a better looking eye with a Thiersch graft than with anything else you can use. I have used lip membrane for some purposes, but as Dr. Parker says, it is hard to manage. You can take a sharp razor, shave off a thin epithelial graft from the lip and apply that on the eyeball, and it makes a perfect covering, with the exception that it looks a little redder than a Thiersch graft, and for that reason, with the female, it is not so popular.

This operation for ectropion which Dr. Parker has mentioned is one which has bobbed up for a great many years. I think it was first described by me in 1892. I have done it several hundred times. It has also been described by many others. It does not make any difference who thought of it first—no matter what you do, if you keep it to yourself somebody else will think of it in a short time. The essential feature is the stretching of the raw surface so as to allow an extralarge graft to be introduced. It is necessary to undermine the skin while you keep the lid on a firm tension; then draw the lower lid up and fasten it to the eyebrow with sutures, and you get as much room as you want. I will admit, that as far as appearance goes, the Thiersch graft is not quite as good as skin from the patient's upper lid, as advocated by Dr. Wheeler, but it is satisfactory for the average old person, and most of these patients are old and do not care whether the lids are exactly the same tint as before, if the eye does not water. I therefore prefer the Thiersch graft because, as Dr. Parker says, one can always get as much tissue as is required no matter which lid is to be repaired; and the stretching of the sound surface permits so large a flap to be used, that there is no need of sewing the lids together.

DR. WALTER R. PARKER, Detroit, (closing): I have nothing to add to what I have already said in the paper, and I come to the platform merely to correct the impression that Dr. Gifford has, that I claim any priority in the ectropion operation. There is not an original thing in the paper, unless it is the use of vaselin in cutting grafts, and if Dr. Gifford did that before I did, I will be glad to make that correction.

THE SURGERY OF SENILE CATARACT.

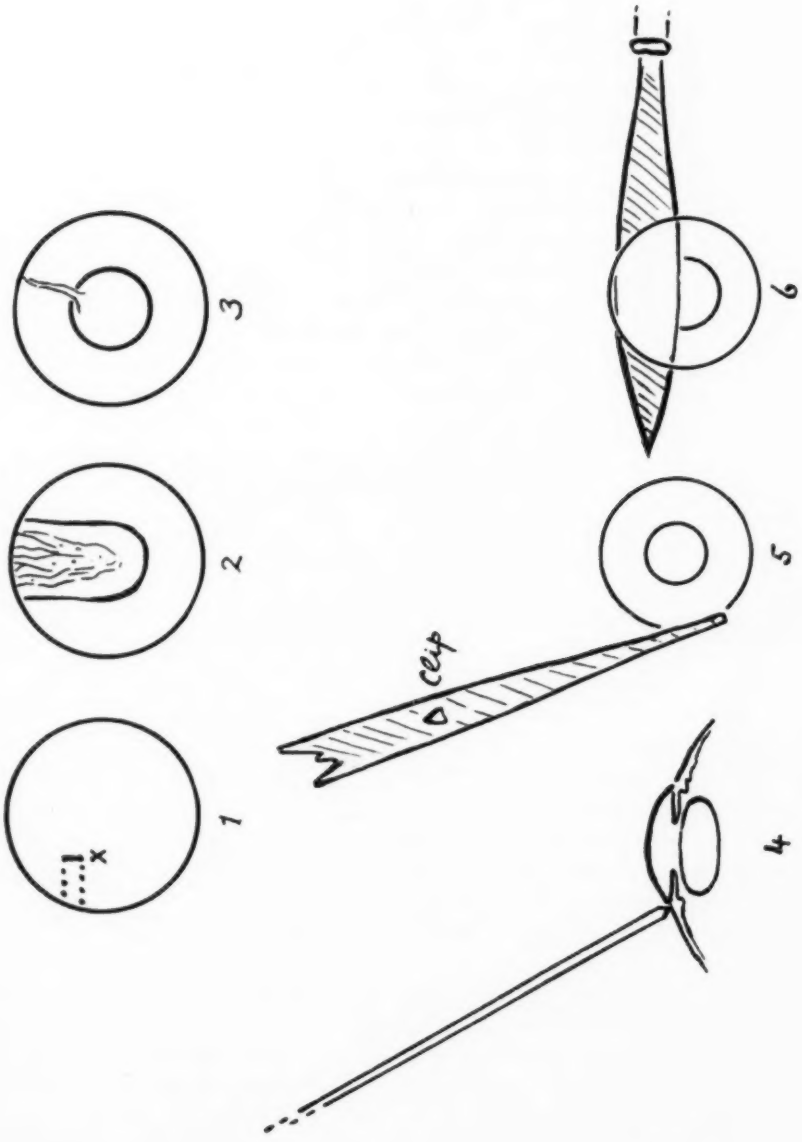
BASIL GRAVES.

LONDON, ENGLAND.

Mr. President, Ladies and Gentlemen: I thank you very much for the honour you have done me by inviting me here to present this subject. As I said yesterday, I find it difficult to express adequately my gratitude for the pleasure you have given me by inviting me to be your guest.

This address was intended in the main to present a few of the already familiar aspects of the subject of cataract extraction in the hope that it is one always worth discussion, and secondly to offer a few suggestions with regard to technic. It was framed on the assumption that it would be open for discussion after it had been read. I did not know until just now that in accordance with the custom of the Academy it will not be discussed. I mention this because I would like to make it clear that I recognize there are many present who have had far more experience than I have had and that in writing this communication my aim has been to seek opinions rather than to impart them.

Any survey of the factors which concern operative technic should include the results of critical observation by modern methods; the precise slit lamp study of postoperative results gives great incentive to, in fact demands, very delicate and precise operative technic. To give an example: A patient may come to a clinic giving the history that elsewhere an extraction of cataract has recently been performed; the vision is poor and the eye is not doing too well and critical observation by the slit lamp may reveal a train of circumstances as clearly as if the operation had been witnessed: e. g. examination of the cornea discloses a faint track (Fig. 1) near the periphery up and out, which gradually inclines deeper and deeper towards a fine scar, x, situated on the endothelial face, not at the periphery, but well away from the edge of the cornea. This shows that the operator allowed the eye to rotate towards the nose as he was passing the Graefe knife into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the cornea, so that its point did not enter the anterior chamber until it had passed obliquely for some way, on the flat, thru the cornea. No evidence of the counterpuncture is found so this is probably where it should be—well at the periphery on the nasal side. Next, when the anterior chamber is examined fibrils of vitreous structure can be traced passing upward from the pupil and permanently adherent to the region of the healed section above (Fig. 2). The train of circumstances is clear: the operator, in trying to press the lens out thru a badly made too narrow corneal section, has forced vitreous out with it.

I once saw a patient who had raised tension following cataract extraction and alleged aftercapsulotomy. The extraction had been a simple one, i. e. without iridectomy. Slit lamp observation revealed a tag of lens capsule (Fig. 3) coming thru the pupil and passing upward over the front of the iris to terminate in an adhesion to the periphery of the anterior chamber above. As it was a simple extraction without iridectomy I was puzzled to know how this could have happened until I learned that it was the operator's custom to deal with a tough capsule some months after the extraction by pulling it out with forceps thru a keratome incision above, instead of needling or cutting it. The use of any form of scissors introduced into the anterior chamber to cut a capsule may lead to the same result.

Another rare cause of high tension, easily detected by critical slit lamp observation, is from the face of the iris having become adherent to the sticky back of the recently healed corneoscleral wound, not at the time of the cataract extraction but later, when the aftercapsulotomy has been improperly done in a manner to let all the aqueous fluid out and so permit contact of the iris with the back of the cornea to which it then adheres.

I propose not to include in these remarks the subject of intracapsular extraction, other than to suggest that on the whole it would seem to be inferior to extracapsular extraction for the one main reason that it is less safe owing to the greater occurrence of vitreous loss. An eye from which vitreous has been lost, even if the loss be only very slight, is thereafter in an unsafe state. Loss of vitreous at the time of the operation means that the fibrillar structure of the vitreous forms an adhesion in the corneoscleral wound, interfering at

first with its healing and in all probability rendering that healing less secure so that a potential track long remains to give access to some low degree of infection; an eye from which vitreous has been lost is never free of the risk of subsequent degenerative changes. Now against this risk, seemingly inseparable from intracapsular extraction, should be contrasted the great relative safety of a properly performed extracapsular extaction which, moreover, yields visual results in every way as good. Some believe that retained cortex causes iritis; I doubt this is often so. However, there is no need to leave cortex in if, in the first place, the lens is correctly delivered and, in the second place, the irrigator is used— a procedure which is quite safe if the previous steps of the operation have so been performed that the posterior lens capsule and the zonular fibers have been left intact. The argument against extracapsular extraction on the ground of its requiring a subsequent needling operation is surely without weight. The needling operation, tho I do not belittle the great importance of its correct technic, is one to which neither the patient nor surgeon need object, and the operation of cutting the toughest aftercapsule is rendered simple by the choice of Wheeler's method. For those who favor the more established and, if I may suggest, stabler method of extraction, viz. the extracapsular, the next main consideration is whether it shall be performed with or without iridectomy. It generally would be agreed that the ideal result is given by a successful simple extraction, i. e. one without iridectomy, and this for two main reasons: first, the intact pupil is a safeguard, acting as a barrier against access and consequent entanglement of lens capsule to the healing corneoscleral wound; secondly, the intact pupil protects the patient from the inconvenience of light glare, a matter of some importance in a sunny country. There are certain other points in favour of simple extraction, which do not carry so much weight as those just quoted, e. g. such as concern final visual and cosmetic results. I believe that, apart from the effect of glare in bright lights, visual acuity is not much impaired by a gap in the pupil edge caused by an iridectomy above; and pleasing as it is for the expert surgeon to see the after results of an accurately performed simple extraction without iridectomy, usually no one else is really interested in the cosmetic disfigurement of an iridectomy: the patient is not and the medical profession in general is not. I

recently saw an eye which, from the standpoint of refined intraocular surgery, was a gross disfigurement: a mutilated iris was badly drawn up into a distorted healed corneal wound, but the eye was quiet and the pleased patient got as much visual acuity as he deemed due. I think that cosmetic and finer visual acuity effects may be grouped as subsidiary in a discussion of the more essential considerations which weigh in favour of extraction without iridectomy, viz. the avoidance of entanglement of lens capsule in the wound, and of light glare.

What are to be considered the main disadvantages of a simple extraction? It will doubtless be agreed that the outstanding, if only, one is the risk, within a few days of the operation, of iris prolapse—with the consequent risk for the patient and inconvenience for both patient and operator. Presumably, but for this risk of prolapse, most surgeons would be in unanimous agreement on the advantage of simple extraction without iridectomy. I know that certain other considerations, which, however, seem minor ones in all but special cases, are sometimes brought forward by those who favor a preliminary iridectomy, such as greater ease, subsequently, in making the section and the capsulotomy, the advantage of a preliminary test operation on the patient, etc.; and tho these are of minor importance as compared with the question of iris prolapse they are worth brief consideration. The section is, aptly enough, often said to be the most important part of the operation. I think that a simple Graefe section, without preliminary iridectomy, is never difficult to perform with certainty of success, even for a skillful novice, if certain points of technic are adopted: First, it is easier to make a good section if the pupil has first been well dilated with a mydriatic. Secondly, the whole procedure is apt to be much more refined if the fixation forceps used are those having a clip which locks their grip: there is prejudice in England against the use of these forceps, in spite of the fact that their grip on the conjunctiva may be released with almost as great ease as that of forceps having no such locking grip, and it was not until the great advantage of clip forceps was demonstrated to me in Dr. Wheeler's clinic that I appreciated their significance. When the eye is held by fixation forceps having no clip the operator has to grip them firmly in order that they shall hold the conjunctiva, and in order to grip them firmly he has to hold them

standing out radially from the surface of the globe where they grip it (Fig. 4): now, he must, while exerting this relatively strong grip, most delicately adjust the amount of pressure which he makes on the globe with the forceps, or for that matter make no pressure at all. This required delicacy in fixation affords one of the first pitfalls for a nervous beginner who, while having to grip the forceps firmly, is apt to press heavily on the globe, when making the section, with disastrous results. If the forceps are clipped automatically to the globe they can be held with the greatest of delicacy, much as a pen or pencil is held, and lying in a convenient and proper direction obliquely, and at a tangent, up across the globe (Fig. 5). Difficulty or danger in the use of the fixation forceps being thus set aside, the use of the knife may next be considered. I assume that most operators prefer to create a conjunctival flap attached to the lower lip of the wound. When the section is made reasonably far back, towards the sclera, the knife in cutting out usually raises this flap, especially if the conjunctival tissue has been elevated by a preliminary small injection of, e.g., cocain solution. It is a matter for consideration as to whether it is worth while dissecting up this flap, much as is done for a trephining operation, before the section is made. This certainly ensures a good flap and consequent quick healing, but it has a few slight objections: unless an assistant holds the flap up while the operator is making the section the sides of the flap are apt to be cut by the knife, leaving an odd tag or two of conjunctival tissue which the operator must be careful to exclude from inclusion between the lips of the wound when the operation is finished; the hanging of such a flap down over the upper part of the cornea may tend to obstruct the view of the top of the anterior chamber when the section is being made. I have not done enough cases of a preliminary flap dissection to enable me to say whether or not I like this procedure for ordinary cases of senile cataract, but it certainly does give an assurance of quick union of the wound afterwards; it has an obvious value in an operation for delivery of a dislocated lens in that it allows of the use of a stitch. The main reason that led me to try this arose from a desire to secure a conjunctival flap while at the same time making a section which was situated well forward, practically in the cornea, rather than far back toward the sclera as must be done if the flap is to be created in mak-

ing the Graefe section: and my aim in making a corneal rather than a scleral section arose from an impression, unconfirmed, that when the extraction is simple, without iridectomy, the tendency to after prolapse of iris may be less when the section is well forward in the cornea. It is well known that those who practice preliminary iridectomy often find that it tends to interfere with the easy making of a complete conjunctival flap with the Graefe knife, owing to the scar left by the use of the keratome in the previous operation: I have not tried it in such a case, but it would seem worth while trying the preliminary dissection of a complete conjunctival flap before making the Graefe section in cases in which a preliminary iridectomy has previously been performed.

As regards the Graefe section, it is very much easier to avoid cutting the iris if the pupil has been well dilated by a mydriatic. Under such circumstances, if the knife is manipulated correctly and the fixation forceps do not press on the globe, the iris has no tendency to get in front of the knife. If, however, it does so I would like to emphasize a procedure I have tried, viz. to withdraw the knife instead of cutting on and to complete the section by using Stevens scissors. To teach the proper use of these scissors, viz. the insertion, without any stress, of one blade into the angle of the anterior chamber —either for completing an unfinished section or for extending one which is too short—gives to the beginner in particular a comforting assurance of a ready means of egress from a difficulty which, in the absence of such provision, often causes him much apprehension. I knew a beginner who was really a good operator, but the making of a Graefe section which he had not the opportunity to practice too often was a procedure he much feared: but his confidence was soon regained when he took to the use of the clip fixation forceps, and also when it was suggested to him not to attempt to make a complete and wide section with a knife but, instead, to make only a short section across the top and then to enlarge it slightly at each side by the use of the Stevens scissors. After doing a few cases successfully thus he soon gained confidence and he afterwards had no hesitation in making a successful and complete section with the Graefe knife. I have known it said that scissors used thus do not make a clean cut but bruise the tissue, with consequent delay in closure of the wound: I am sure that this is not so and that a wound heals as quickly after the proper use of scissors.

A not uncommon practice in some clinics is to open the lens capsule with the point of the Graefe knife as it is passed across the anterior chamber in making the corneoscleral section. It of course looks dexterous. The "excuse" which is usually advanced in favor of this procedure is that one less instrument is thereby introduced into the eye. The risk of infection resulting from the introduction of one extra instrument into the eye is practically nil when the operator works with dry instruments and dry hands off which no drops may gravitate to carry organisms down to the instrument point which enters the eye; whereas, on the other hand, the mishaps which result from the practice of making a cystotomy with the knife when it is half way thru the section are by no means infrequent in the hands of even the most skillful of operators. Many considerations should bar this procedure: the knife has to be tilted out of its straight path, an adequate objection alone. I have known, admittedly only occasionally, the cut lens capsule to remain caught on the end of the knife and to pull the pupil edge sideways with resulting complications when the point travelled on to make the counterpuncture; but the main and surely emphatic argument against this procedure is that an eye opened by a Graefe section may be considered to be in a comparatively safe state until the lens capsule has been opened, provided the section has been made without any stress on the globe such as to cause rupture of the zonular fibers. After the capsulotomy comes the anxious time: it is one thing to complete successfully a simple Graefe section on a patient who lacks control but quite a different thing to do so on the same patient when the lens capsule has been opened; if the use of scissors is for any reason desired in the making or completing of the Graefe section, then their employment is safe and simple before the capsulotomy but very difficult after it. I can quote a curious incident: In doing a simple extraction I had passed the knife across the eye, i.e. puncture and counterpuncture had been made, but I could not make the knife cut out; I withdrew the knife and on examining it found that, tho its point was intact, its edge almost thruout had been turned, evidently from an accident in the sterilizing. I dissected down a conjunctival flap from above to the limbus and then, with Stevens scissors, completed the section beneath this flap from puncture to counterpuncture, without in any way injuring the iris, and this could

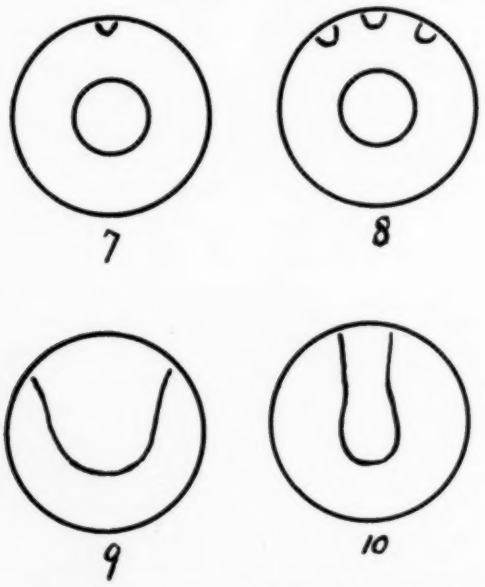
be done with a deliberate assurance and ease which would have been impossible had the capsulotomy already been performed by the Graefe knife. Some say that the use of a cystitome in the presence of an intact iris is difficult: this is hardly so if the proper instrument is properly used. The occurrence of blood accumulating in the anterior chamber, from a bleeding conjunctival vessel, is sometimes urged in support of the cystitome being a difficult instrument to use: if this happens it is simple—the lens capsule being unopened—to pause, use adrenalin drops and wash the blood out of the anterior chamber with an irrigator which admittedly must have a reasonably large nozzle yielding a good flow, aided, if necessary, by gentle stroking of the corneal surface.

Before the subject of making the Graefe section is left two more points are worth consideration. It occurred to me that the lips of the wound would lie in more intimate apposition, and healing would consequently be quicker and ultimate astigmatism less, if the entire cut could be made while the globe retained its normal shape: as is well known, it does not in the latter period of the ordinary Graefe section. I tried various ways to effect this: one was to use a specially made very broad knife for the section—like a Graefe knife save that it was much broader—so that puncture and counterpuncture being made with no escape of aqueous fluid little cutting motion remained to be done after this broad knife had transfixed the anterior chamber (Fig. 6); but I found that while the knife entered the eye easily enough against the unbroken ocular tension, the counterpuncture was by no means easy be cause of the comparatively blunt angle of the end of this broad knife. It may have been that this difficulty would not have existed had the particular knife been made with a more tapered extremity, but I did not carry the trial further. I have tried other means of making the section so that it might be completed while the globe is in its normal spherical shape, including cutting from above downward; also using a keratome—either one centrally above, the cut being afterwards enlarged laterally with Stevens scissors, or two, i.e. one on each side so as to make two cuts simultaneously which would afterwards be united by scissors. I think that none of these procedures is advantageous and that a simply made Graefe sec tion, whether the iris is intact or whether there has been a preliminary iridectomy, cannot be beaten.

I have referred to the greater ease with which the section may be made, when the iris is intact, if the pupil is well dilated with a mydriatic. Now the two well known causes of the iris getting in front of the knife edge are, first, pressure on the globe with the fixation forceps and, secondly, incorrect manipulation of the knife so that the aqueous fluid is allowed to escape too soon. If the iris once gets in front of the knife edge, unless the operator is going to withdraw the knife and complete the section with scissors, the only maneuvre left as an attempted means out of the difficulty is to lift both the knife and the fixation forceps so as to put an upward stress on th front part of the globe. The necessity to do this may not always be the fault of the operator; had behavior on the part of the patient may cause these circumstances, tho this will happen but rarely if the local anesthesia is good, the orbicularis paralyzed by novocain and the patient properly quieted and reassured. However, it is worth while considering that maneuvres of this sort, involving stress on the eye while the section is being made, beside having the objection that the globe while being cut is distorted from its normal spherical shape, may lead to rupture of the zonular fibers, with consequent predisposition to escape of vitreous fluid when the lens is being expressed. In this connection I have a suggestion which I have never yet tried in practice. In the Transactions of the American Ophthalmological Society for 1925 is an account of some observations on the state of tension of the lens capsules under the influence of various drugs, in which it will be seen that the effect of cocain and of mydriatics generally is to put tension on the lens capsules. I found that when the lens capsules are tense under the influence of homatropin and cocain the instillation of one drop of 1% eserin sulphat begins, within two minutes, to cause a relaxation of the lens capsules which becomes extreme within five minutes, but no contraction of the dilated pupil begins until after an interval of some nine minutes. It might be worth while using this to bring about relaxation of the zonular fibers just before the section is made: thus, the pupil having been dilated with homatropin and cocain, the operator, when ready to make the section, would instill a drop of 1% eserin sulphat just two minutes before and would then make the section before the eserin could influence the size of the dilated pupil; in all probability, under these circumstances, any distortion of the globe

during its sectioning would not cause rupture of the zonular fibers. For those who wish to perform intracapsular extractions the effect of this procedure might be worth trial, in order to see whether removal of the lens might be favored by relaxation of the zonular fibers: but I do not suggest it on the theory that it would be, tho I think that the laxer anterior capsule could be more easily gripped and that the preliminary manipulation into a position for dislocation might be favored.

Supposing an accomplished operator considers that he can make a section as well when the iris is intact as when a preliminary iridectomy has been performed, it is the question of



the danger of after prolapse of iris which will weigh most with him in deciding as to whether he will leave the iris intact or will perform an iridectomy when he operates for the extraction; if, however, he believes in the advantage of the intact pupil as a means of keeping lens capsule out of the wound he may consider the procedure long ago adopted by Chandler of Boston of making a small peripheral buttonhole iridectomy (Fig. 7). Clinical slit lamp observation of patients who have been operated on for cataract, as well as the study of laboratory sections, cannot but emphasize the seriousness of inclusion of lens capsule in the healing corneoscleral wound and the great advantage of the pupil being left intact in order to

prevent this serious, if rare, complication. (In connection with this, consideration should be given to the view of those who assert that if a piece of capsule is removed with capsule forceps, entanglement of capsule in the wound will not occur even in the presence of a complete iridectomy.) We know that if the iris is left intact after prolapse will inevitably occur in a certain percentage of cases. The peripheral buttonhole iridectomy is much favored by some operators in England and I think I have had a fair opportunity, over years, of observing its results: my impression is that while it tends to guard against prolapse it is by no means a guarantee, because the intact region of the iris, on either side of the buttonhole and especially near the extremities of the section, not uncommonly prolapses. So I tried a procedure, which is an extension of this principle, of making three little buttonholes, first one in the center, and then one near each extremity of the section (Fig. 8). I have done ten cases in this manner and in no case has there been any prolapse.

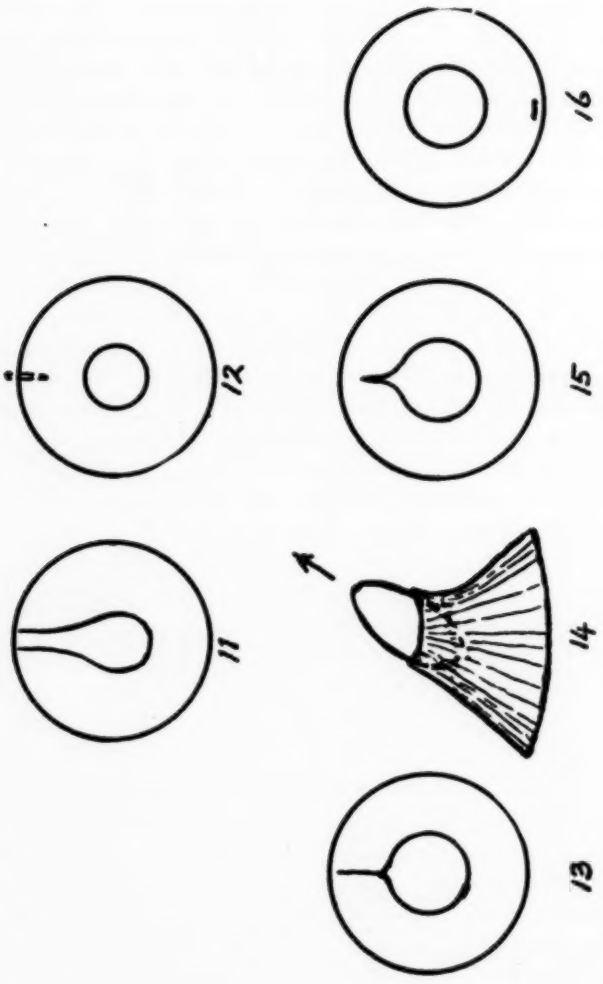
I suggest that the probable mechanism of after prolapse of an intact iris is this: When the eye is dressed immediately after the operation there is no anterior chamber and the iris lies in intimate contact with the back of the cornea; soon the secretion of aqueous fluid by the ciliary body leads to the formation of a chamber because at first the iris retains its intimate contract with the back of the cornea, acting as a barrier against the leakage of aqueous fluid thru the unhealed wound. If at this stage the patient puts any sudden strain on the eye, as by a cough, the aqueous fluid collected behind the iris forces the iris out thru the linear wound. If, however, there is a hole in the iris right opposite the wound, the fluid will not collect in this way behind the iris but will leak out thru the wound until the lips have united. But it would seem that the one small buttonhole situated opposite the center of the wound is often inadequate to prevent prolapse probably because the lips of the wound may, conceivably, chance to unite opposite the buttonhole before doing so on one or both sides of it, and under such circumstances the aqueous fluid, thus allowed to accumulate, may force iris out thru the unclosed end or ends of the wound. Hence the presence of more than one peripheral buttonhole behind the wound, say two or three in all, affords a better chance of permitting a more prolonged escape of aqueous fluid and a consequently longer

time for closure of the wound uniformly thruout its length before any anterior chamber can be formed.

I know that the ten cases which I have done are not enough to test the efficacy of the method as a guarantee against prolapse, but two of these behaved, after the operation, in a way which certainly tends to afford evidence in support of its value: they were two ladies, sisters, on whom I operated on the same day; both were ideal patients with perfect selfcontrol and steadiness, but the one vomited several times and also sneezed during the night following, and the day after, the operation, and her anterior chamber did not reform till the third day. About the fifth day she rubbed her eye in her sleep and in the morning the anterior chamber was found to be flat again, but it reformed in the ensuing 24 hours. There was no prolapse of iris at any time nor did the face of the iris become adherent to the corneoscleral wound. The other sister gave the eye a blow or rubbing, in her sleep during the fourth night, causing hyphema and collapse of what had been a fully formed anterior chamber since the day after the extraction: the anterior chamber subsequently became reformed—it should be mentioned that the conjunctival flap had held—without any union between iris and corneoscleral wound. In so far as a judgment can be formed on the basis of a few cases I am impressed with the efficacy of this procedure as a guard against iris prolapse; where the previous stages of the operation have been satisfactorily and delicately performed so as to leave an intact zonule and intact posterior lens capsule, and if, as is essential, the iris has been properly anesthetized by a small preliminary subconjunctival cocain (not novocain) injection, and if an assistant lifts the weight of the speculum off the globe, in most cases there is not need for undue haste to remove the speculum and close the eye, and the procedure of making these three little peripheral buttonholes iridectomies in the final stage of the operation is simple and safe. Of course, it is undesirable in certain cases; tho straightforward, the procedure is delicate and calls for absence of anxiety on the part of both patient and operator. If the patient lacks selfcontrol, or if a definite tendency to prolapse is indicated by a pear shaped pupil, such circumstances may indicate that it is better to complete the operation by making one simple complete iridectomy rather than to persist in adhering to previous intentions in the face of indications to the con-

trary. I have used ordinary iris forceps for making the peripheral buttonholes; the value of the Hess iris forceps, the teeth of which have a backward inclination at an angle to the terminal part of the blades, is questionable: if these teeth are too long and too sharp there may be risk of injuring the zonule. Sometimes the teeth of these Hess forceps, besides being too long are made so very thin and sharp that, while they will readily perforate the iris, they will not grip and lift it. Another objection to the Hess forceps is that they are commonly made much too small: delicacy in the use of an instrument is not proportionate to its reduction in size. The procedure is delicate and de Wecker's scissors are desirable rather than ordinary large iris scissors. I have in all my cases made the central hole first and before this is done the iris, if it is prolapsed, must be replaced in its normal position by means of the repositor or irrigator: then after the central hole is made a slight stroke on the outer surface of the cornea will replace the iris in its normal position for the making of the second hole, this replacement, if it is not spontaneous, being repeated before the third and final hole is made. I had been doing this operation some little time before, at the Oxford Ophthalmological Congress recently, I heard Colonel Smith say that he has been making such multiple peripheral iridectomies in his cases of intracapsular extraction, but I understood him to say that he makes the multiple buttonholes before he expresses the lens in its capsule. I cannot speak from a knowledge of this procedure in cases of intracapsular extraction; but when a lens nucleus only is extracted I would think that the making of the peripheral iridectomies before the nucleus is delivered might tend to complications in that they might be inclined to tear under the stress of its delivery.

It is a not uncommon experience that when a peripheral iridectomy has been accidentally performed by the Graefe knife in the making of the section the lens, when being delivered, may show a tendency to force itself thru the iridectomy instead of thru the pupil, and I think that most operators, recognizing this, meet the emergency when it occurs by completely cutting the iris right thru to the pupil before they express the lens nucleus from the eye. However, a peripheral iridectomy accidentally made in this manner is usually much more extensive than the small deliberately performed buttonhole iridectomy; I have not yet tried it but it would seem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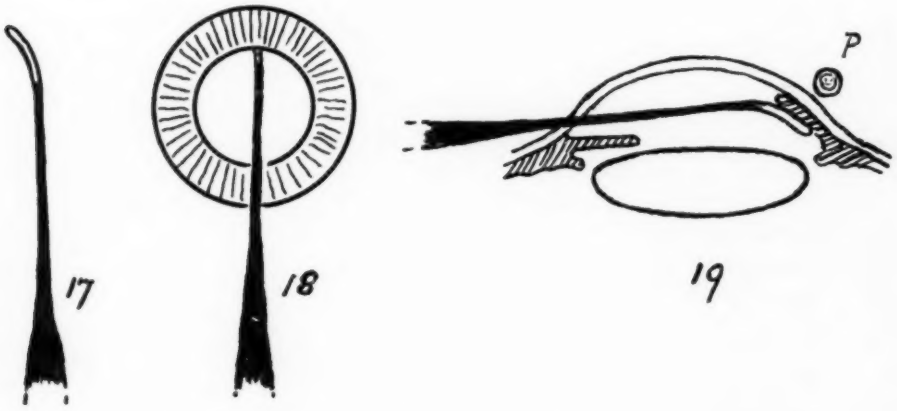
worth while trying the making of the three buttonhole iridectomies before the capsulotomy is done, tho I feel that they can as easily be made after the lens nucleus is expressed and with practically no more danger provided, as suggested above, certain conditions obtain chief of which is that the previous stages of the operation have so been performed that the zonule and posterior lens capsule are intact, the risk of vitreous loss being therefore minimal. In the rare cases, probably when the eye is prominent and the lid pressure is somewhat posterior, in which the lens nucleus expresses itself spontaneously immediately the cystotomy is made, this should be taken as an indication that the contents in general tend to protrude and that the iris will be likely to become prolapsed unless a complete iridectomy is made; and altho it may be disappointing to depart from a defined procedure which was the initial aim, it is better judgment to submit to such warning and to make a complete iridectomy. This operation of a simple extraction of the nucleus combined with multiple peripheral buttonhole iridectomies seems to me a very satisfactory one and I intend to give it fuller trial. But I would like to give a brief account of another way in which I have tried to prevent iris prolapse.

Unless it is considered desirable in order to make the performance of the Graefe section easier, the only other main object of a preliminary iridectomy, done at a previous operation, is the prevention of after prolapse of iris. I learned in this country that it is not necessary, in order to prevent iris prolapse, to make an extensive iridectomy (Fig. 9), and that a very narrow complete iridectomy suffices (Fig. 10), the pillars of such an iridectomy showing no tendency to prolapse thru the much wider Graefe section. The essential points in the technic for making narrow preliminary iridectomies depend on the manner of using the keratome, of gripping the iris and of cutting the iris, so that it is possible to make always the narrow keyhole pattern of iridectomy, having straight almost parallel pillars. It occurred to me that if a preliminary iridectomy is desired even narrower still, i.e. slit like (Fig. 11), it could be made by access to the iris thru a limbal cut which is at right angles to the limbus (Fig. 12) instead of concentric with it. Obviously the keratome is not suitable for making such a cut, but the cut can be made by using a Graefe knife with its back directed towards the surface of the globe. I have done only one case in this way and it gave a very satisfactory narrow iridectomy—the type of iridectomy commonly

resulting from its performance thru a trephine hole. If it seems worth while pursuing this operation I have a plan for a small cutting instrument with which it would be easier to make the radial cut in the globe than with a Graefe knife.

The next consideration which occurred was whether there need be an iridectomy at all but whether, instead, a radial iridotomy would suffice, i.e. a radial cut in the iris without excision of any iris tissue (Fig. 13). The aim was this: when the lens nucleus is expressed thru the pupil, not only is the sphincter of the iris stretched and dilated to admit the passage of the nucleus, but the mere fact that the iris is anchored at its circumference where it is attached to the globe means that forced expression thru the pupil also stretches the iris radially (Fig. 14), i.e. stretches its dilator fibers, much as the membrane of a drum is stretched radially in all directions if pressure is put upon its center. It seemed reasonable to suppose that this stretching of both sphincter and dilator fibers—already none too strong in old people—must cause atony, and that after prolapse of iris may to some extent be contributed to by its musculature being thus atonic. I confess that I doubt this factor of atony is of paramount importance, save in so far as it would render ineffectual the action of atropin, because if a semidilated iris is atonic or not it will probably prolapse thru an unhealed wound if, under sudden stress, it is subjected to pressure from behind by a collection of aqueous fluid for which no egress is provided by a hole or holes in the iris. However, these considerations led me to devise a procedure which would bring about a more effective dilatation of the pupil, viz. by cutting the sphincter muscle radially (Fig. 15) as a preliminary operation, so that when a mydriatic were afterwards given the unopposed dilator fibers could act to such advantage that the well dilated iris would, all round, practically be tucked away tight up against its own base. I have now done some few cases of this preliminary operation and without going into the full details that have transpired—and they are many—I will just indicate some of the points of interest. The principle is to cut radially the iris above, by introducing thru a small hole, made in the corneal periphery below (Fig. 16), a tiny curved cutting instrument whose sharp edge is convex, having a curvature which corresponds approximately with that of the back face of the periphery of the cornea: the simplest way to describe the knife is to say that it is rather like a Ziegler needle in shape and size, but its end

is blunt and it is the convex and not the concave edge which is sharp (Fig. 17). I use an ordinary two edged fine capsulotomy needle to make a small puncture just thru the corneal periphery below, without losing any aqueous fluid. The curved instrument is then introduced thru the puncture and is passed upward across the anterior chamber with the flat of the blade lying parallel to the face of the lens; its extremity is passed behind the already dilated pupil above, between lens and iris (Fig. 18). To effect this it is necessary to let the flat of the blade lie deliberately on the lens face because if it does not the end of the instrument may, instead of passing behind the iris, engage the pupil edge which is quite thick in some persons. When the instrument has been passed into this posi-



tion it is rotated till its sharp convex edge looks towards the cornea and the handle is then depressed until the sharp edge comes up against the back of the peripheral part of the cornea, with the iris interposed between it and the cornea (Fig. 19) Up to now the operator has been fixing the eye by gripping it near the limbus above, but he now discards the clip fixation forceps—the shank of the instrument which passes thru the corneal aperture providing sufficient fixation—and he then presses on the corneal surface, from without, over the region where he has aimed to cut the iris. I find that a convenient instrument to use for this surface pressure is a thick nasal duct probe (Fig. 19, p); the pressure must be enough to produce the required complete radial cut in the iris, but insufficient to produce a deep cut into the cornea. As a matter of fact con-

siderable pressure may be exerted, from without, down on to the cutting instrument without fear of its deeply cutting the back face of the cornea, because the cutting instrument is, or should be, stationary on the corneal face and should make no sliding cutting motion. I may say that there are many small points of technic about this operation which it may be advantageous for anyone who is trying it to know, but I will not take time now in mentioning more than one or two of them. The iris must have been properly anesthetized by subconjunctival cocain* (not novocain) injection. I have, here, the two knives I have used for the cases which I have so far done: the main points in their design concern the length and curvature of the flat cutting part and also the diameter of the shank which should, tho loosely, fill the hole of entry so that aqueous fluid is not lost during the passage of the instrument across the anterior chamber. I am undecided as yet whether it is better to dilate the pupil fully by the previous use of atropin or only partially, as by cocain. If the pupil is well dilated the deliberate letting out of some aqueous fluid, when the knife is in position, will reduce the size of the pupil, and this maneuvre can be used to bring the pupil and iris more completely over the end of the knife. There are two factors in particular which I would like to speak of now. First, if the cornea of these cases is afterwards examined with the slit lamp it reveals a clear linear cut of the endothelium on the back face of Descemet's membrane, and perhaps partly into Descemet's membrane, but I have never seen an opacity result from this cut, in spite of the clinical teaching that a wound in Descemet's membrane leads to an overlying opacity by letting aqueous fluid gain access to the cornea. It may be that the cut is never deep enough; but should a small local faint opacity result at the periphery of the cornea above it would not matter.

The second point I would speak of now concerns a peculiar after reaction which has occurred in three of the twelve cases I have so far done. The operation is painless when it is performed, being done under subconjunctival cocain injection: but in these three cases a reaction, similar in all three, started about two to three hours after the operation: it was characterized by intense pain, much more severe than is encountered

*A practice so common in many eye clinics in the United States. A minim or two of 1% or 2% cocain solution suffices. Novocain, even tho fresh, does not give such good anesthesia of iris.

when the effects of the local anesthetic pass off after any ordinary operation on the iris. So great was the pain that it was difficult to induce the patient to open the lids which were highly sensitive, tending to be swollen, so that the patient could not tolerate their being touched. When I first saw this I was alarmed, but when with difficulty I succeeded in getting the lids slightly opened I found a quiet looking globe instead of the injected one I had expected to see. Anyone accustomed to postoperative care of eyes would expect to find a very red eye in a patient suffering such pain after an intraocular operation and the reaction I am describing is quite unlike any postoperative one which I have seen: nor have I ever seen it after accidental injuries to the eye. The severe pain was accompanied by much lacrimation; every voluntary attempt on the part of the patient to open the eye caused a severe stabbing intensification of the pain, so that he would fear even to make the attempt. It might be asked whether in doing these cases I had allowed the blunt end of the instrument to pass so far in behind the iris as to injure the ciliary body: I know that in no case did this happen; moreover, injuries to the ciliary body have a sufficiently recognized after course. Simple cutting of the iris, followed by the use of atropin, causes no such reaction; nor does a surface cut or a complete penetration of the cornea have this effect. It would seem that the pressing of the cornea against a stationary sharp edge applied to its back face in some way leads to this after reaction which must comprise a very strong stimulus of the fifth nerve endings resulting in the severe pain, lacrimation and reflex spasm of the orbicularis. It might be suggested that the cut involves one of the nerve trunks which traverse the cornea near its periphery; but these nerves, as is well recognized from slit lamp work, seldom lie deeper than the anterior half of the corneal thickness and never deeper than the anterior two-thirds, while slit lamp observation of the cases I have so far operated on shows that the clear linear cut on the back face of the cornea extends to only a very slight depth. It may be that the effect of the cut is to cause a transient edema of the corneal substance between unyielding uncut limiting membranes, with resulting pressure on the nerve in the vicinity. Subsequent slit lamp examination reveals no abnormality of the nerves or of the corneal substance over the line on the back face which represents the place where the sharp edge was applied. The only

clinical condition in which the sensory reaction somewhat resembles this now described is that occasionally seen in severe cases of acute interstitial keratitis and it is interesting to consider that in this affection the corneal changes are usually most pronounced at or near the deep face.

In each of the three cases in which this reaction occurred a narcotic was given and the pain had passed off by next morning; but tendency to lacrimation on exposure to strong light lasted for a long time. It should be noticed that in appearance the eyes became in no way flushed and careful slit lamp examination revealed no abnormality such as corneal precipitates or increased visibility of the aqueous beam. It seems clear that the sensory reaction, whatever its explanation, is unassociated with any inflammatory reaction or for that matter with any reaction of the clinical type which we are accustomed to regard as dangerous; there has been no sign of iritis afterwards and the iris cut, once made, is permanent in accordance with the habit that a normal iris in normal aqueous fluid does not form granulation tissue.

If the percentage of cases in which this reaction occurs is high I think it should rightly be considered as a contraindication to this suggested operation as a preliminary procedure to cataract extraction on account of the discomfort to the patient, tho the operation may have its place in certain other conditions where it is desired to make an iridectomy without having to pull the iris out of the eye in order to do so. Another consideration on which I will not dwell here is that when a cut has been made in the endothelial face of the cornea the process of repair is slow and until the repair is complete the healing cut is sticky. If a subsequent operation is done which involves letting out aqueous fluid, e.g. lens extraction, it is possible for the lens capsule to gain adhesion to the still sticky cut while the anterior chamber is collapsed. The maximum period for which such a cut can remain sticky has, as far as I am aware, never been discussed: but it certainly is long and may amount to several weeks.

I had hoped to carry my investigations much further by this time, but circumstances have prevented me and this report on this procedure is therefore premature; in fact I am unable at present to give conclusions as to the value of the procedure in preventing prolapse of iris after the subsequent extraction of the cataract, which was the object of the operation, and I must defer this till a later report.

A communication on the technic of cataract extraction to be complete should deal with the capsulotomy, the use of capsule forceps, expression, the removal of cortex, and after treatment, besides other considerations, all of which would occupy too much time. I would just say that slit lamp observation leads me to favor a capsulotomy done in the form of one simple vertical straight cut. For irrigation, the Noyes irrigator on account of its compactness and ease of sterilization is more convenient than the pattern which is fed thru a tube from an overhead container: but the tip should not be made so fine as to prevent the passage of an adequate flow of fluid to provide the flushing action necessary for displacing the cortex.

I would like to say a word about Wheeler's method of aftercapsulotomy, which seems so valuable because no operator who practices it need fear difficulty in dealing with either a thin or a tough membrane. A strong point in connection with this operation is that if it is properly done not a drop of aqueous fluid is lost so that there is no collapse of the anterior chamber even for a short time after the operation: as the result of this the vitreous, tho cut, does not come forward thru the pupil—as I have ascertained by slit lamp study of these cases after operating on them—and it certainly never comes forward sufficiently to gain connection with the small corneal penetration.

In concluding this paper I would like to urge the view that the relatively greater danger of intracapsular extraction lies in the risk of vitreous loss which should be regarded as a serious surgical accident even if the loss is only slight: whereas extraction by opening the anterior capsule is a well established procedure in which the risk of such a mishap is reduced to a minimum in the hands of an operator who proceeds so as to preserve intact the posterior capsule and the zonular fibers; that the necessity to perform the subsequent minor operation of capsulotomy is no offset in support of intracapsular extraction, especially since we have a method of capsulotomy which affords a certain, easy and delicate way of dealing with the toughest capsule; and finally that it is desirable, circumstances being favorable, so to perform the extraction that the patient is left with an intact pupil provided that the operator can select some technic which, in his opinion, affords a reasonably sure guarantee against the risk of after prolapse of iris.

THE ANOMALOUS FORMS OF TUBERCULOSIS.

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To understand a disease such as tuberculosis, it is almost necessary to regard the causative bacillus as possessing reasoning power. The parasite must gain access to the host, and in order to prepetuate the species, it must find a means of egress from the host and transit to a new victim. Should death follow infection, the bacillus has in part failed, for a colony is now lost. Physicians, in advocating the specific remedy of rest, aid in converting active disease into a more chronic type, and so abet the parasite by producing an increase of carriers. The best opinion today regards tuberculosis in general as an air borne disease, somewhat similar to pneumonia, and a direct and possibly frequent contact with a consumptive, or carrier, as necessary for infection. Bovine infection is, of course, an exception, but the bovine bacillus is productive of less than one per cent of pulmonary tuberculosis and, according to some estimates, 18% of bone and joint disease. As Theobald Smith has so well said, the human tubercle bacillus is "tuned up" to the human lung. Over the centuries, the tubercle bacillus has acquired the power to produce lung destruction with resulting expectoration, and so the cycle from man to man continues.

The initial focus of tubercle infection is, in this country, almost invariably pulmonary, and the regional tracheobronchial lymph nodes quickly become diseased. Should the tubercle bacillus attack the tonsil which, according to some investigators, it appears to do, evidence of infection having been determined in 2 to 3 per cent of tonsils removed, the organism passes to the cervical nodes and, eventually, by circulatory route, may reach the lungs and tracheobronchial glands. In these glands, the bacillus, according to Cobbett in England, can remain dormant thru the life of the individual. The initial lung and tracheobronchial lymph node infection usually takes place in childhood and, after Bayle, may be termed occult tuberculosis. Some immunity appears to result

from this childhood infection, and the pulmonary tuberculosis of the adult may arise from reinfection from the original lung or lymphatic disease, or from reinfection from contact with a consumptive. In X-ray films of the chest, we so often see increased markings which accentuate some of the branches of the bronchial tree. Such accentuations suggest stasis of blood and lymph and, as Bushnell pointed out, it is in the circumstance of lymph stasis that the tubercle bacillus delights. The infected minute lymph follicles at the bronchial bifurcations may also rekindle. The development of pulmonary tuberculosis in diabetic patients who, thru more careful hygiene, may not be carelessly exposed to consumptives, and the extensions of tubercle into other lobes of those already diseased, would seem to substantiate the reasoning of reinfection as being from within.

The triumph of the bacillus in establishing active pulmonary tuberculosis is usually accompanied by cough and expectoration. Tissues such as those of the larynx and intestines may be exposed to myriads of tubercle bacilli and secondary disease may result. On the other hand, there appear to be some patients with laryngeal and intestinal tuberculosis, who have developed these complications when sputum has been difficult to obtain or few bacilli detected. It may be that infection thru the blood stream can produce laryngeal and intestinal tuberculosis, just as it is by this route that tubercle of such organs and structures as the eye, ear, bone, and joints arise. The effort of the tubercle bacillus, having established itself in new tissues and organs, is to obtain an outlet by sinus formation. Such lesions are anomalous. Similarly in typhoid fever, necrosis of the spine, or of a rib, is not typical of the typhoid life history in which, altho a bacteremia, Peyer's patches and the spleen supply the susceptible tissues.

The anomalous manifestations of tuberculosis, except possibly kidney infection, appear to be in some degree more amenable to therapy such as rest and radiation than are lesions in the lungs. This may be due to the tissue defense being innately more potent and the soil less congenial to the bacillus. It has long been observed by physicians, that the development of an ischiorectal abscess is accompanied by improvement in the tuberculous processes in the lungs. Similar observations have been made following the onset of laryngeal, intestinal, and aural tuberculosis. Tubercle developing in the eye may

have similar results. Such improvement in the lung disease is apt to divert attention from this organ, and the local manifestations alone become subjected to treatment.

Tuberculosis in any part of the body should be studied as a general and constitutional disease. A patient may present himself with a tubercle of the choroid. Such a patient should undergo a careful clinical history and investigation. The taking of a correct history is perhaps the most important part in the diagnosis of tuberculosis. In addition to chest examination, the pulse and temperature must be observed, while the patient is resting, for from one to two weeks. It is not enough to take the temperature at a few office visits, for normal or subnormal readings may be noted as a result of the patient's fatigue, and the actual temperature possibly depressed by too much absorption of tuberculin from his own foci. Should such a patient be discovered to have a slight persistent fever or a continued increase in pulse rate, a rest regime is indicated until stabilization occurs. It would seem to be unwise to institute tuberculin treatment until this time. The last stronghold of tuberculin therapy is in tubercle of the eye. Even the tuberculin treatment of tuberculous adenitis has been abandoned in consequence of the quicker and better results obtained by insolation and X-ray. There can be no doubt that tuberculin treatment of eye lesions has in many instances yielded excellent results, on account of the care and watchfulness of its administration. However, Rollier of Leysin notes, that in numerous instances of eye tuberculosis, tuberculin treatment has failed, but such patients have recovered following sun treatment. "Insolation thru closed lids is very well tolerated by the retina. Treatment consists in exposing the eyes to the sun with the lids closed; this is repeated several times a day for periods of ten to twenty minutes. As might be supposed, it is necessary to use gradual progression in these cases, just as one does in the treatment of other localizations." Apart from the local insolation, Rollier employs also the general sun bath, which must be carefully ordered and controlled. The valuable results obtained by insolation in tuberculous laryngitis are known to all.

The numerous patients who are submitted to tonsillectomy are more and more receiving a general examination before operation, yet I can recall only two patients consigned to my care, in whom operation was postponed because active pulmo-

nary tuberculosis was discovered. On the other hand, in history taking, I find that a large number of patients relate tonsil removal a few months before their real condition is recognized. An X-ray chest examination before tonsils are removed, would be a wise routine procedure.

To touch briefly on the matter of prevention of tuberculosis, it is noted that the mortality has been declining rapidly in the last twenty years. With failure to discover a specific, for vaccination or cure, direct attack has not been possible; but the flank attack, consisting of education, childhood welfare, improved social conditions and the great growth of sanatoria, in which isolation is involved, is bringing not unexpected results. Koch, in his Nobel lecture in 1906, called attention to what partial isolation of infected cases accomplished in the case of leprosy in Norway. "There they have not isolated all the lepers, but only a fraction of them, including, however, the most dangerous; and the result has been that the number of lepers, which in 1856 still amounted to 3,000, has now gone down to 150. This is the example to be followed in the combating of tuberculosis, and if all cases of tuberculosis cannot be provided for, at least as many as possible, including the most dangerous, i. e., those in the last stage of the disease, ought to be lodged in hospital."

The complement fixation test for tuberculosis is of scientific interest but has no practical application, since too many false positive and negative results occur. Speaking generally, it seems to accord somewhat with the course of events, but we abandoned its use after some 1,000 tests. As regards treatment, there is only one remedy of outstanding merit and universal application in the treatment of tuberculosis, and that is rest. So far as I have been able to search medical history, Trousseau in 1837, advised complete rest for laryngeal phthisis, and advised his patients to write on a slate. This is perhaps the first occasion in which rest was advocated for tuberculosis. Carlyle, in 1849, in allegorical phrases, urged his consumptive friend John Sterling to sit still. Some sixteen years ago, a youth of eighteen came under my care with advanced lesions of pulmonary tuberculosis. After a few weeks, all signs and symptoms being greatly improved, I told the lad he might get up from his rest cure. He at once asked what had caused his improvement, and I replied that the rest in bed was responsible. Immediately the next question came,

asking why then should he get up? The patient stayed in bed for eight months after I had given him his liberty, and he made a complete and lasting cure. By specifying that our patients should rest on the side which is actively diseased, by the application of shot bags to the apices of those who have bilateral disease, by artificial pneumothorax and thoracoplasty, we are at last carrying out the principles of rest which have been so successful with our surgical colleagues in treating tuberculosis of bones and joints. Physicians, however, have not yet mastered the necessity of prolonging the rest regime to ensure complete recovery, nor are they skilled in the management, so that the patient does not say with Homer "Too much rest itself becomes a pain."

THE USE OF TUBERCULIN IN OCULAR TUBERCULOSIS.*

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Koch, the discoverer of the tubercle bacillus, was the first to advocate the use of tuberculin in the treatment of tuberculosis. He thought that it was a specific and would cure the disease. His original article, published in 1891,[1] is worthy of careful perusal. Following the publication of the article, tuberculin was taken up with enthusiasm by clinicians, and it was administered to tuberculous patients regardless of the type of case and frequently in excessive doses. The results were so disappointing, that it was soon abandoned by the majority of the medical profession and regarded as both worthless and dangerous. Even today, a strong prejudice against its use exists in the minds of many able physicians. However, some of the early observers felt that the preparation was of some value, and they have investigated its action sufficiently to prove, that when properly administered to suitable patients, favorable results can be obtained, and that the continuation of its use is desirable and justifiable. There is certainly sufficient evidence, that in well selected cases it has a beneficial influence. We must accept the opinion of discriminating observers who have used tuberculin, rather than of those who have never used it and claim that it is dangerous. It is true that tuberculin is no more of a specific than hygiene, diet, etc., but it has its place in certain types of the disease. Its value in ocular tuberculosis, when properly administered, can hardly be disputed. This will be discussed later.

In his experimental studies, Koch found that the injection into tuberculous animals of diluted doses of tubercle bacilli in suspension, or suspensions of dead tubercle bacilli, did not give satisfactory therapeutic results. He then thought, that to get

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results, one must extract from the bacillus an immunizing substance. His first attempt in this direction was the separation of the bacilli from the culture media on which they grew, in the hope that the filtered media would contain the byproducts of the bacteria, and that it might promote immunity when injected into the body. This substance he called Old Tuberculin. Since then, several other preparations have been advocated and used in treatment.

Old Tuberculin, O. T., is prepared from cultures grown on media consisting of 5 per cent glycerin alkalin broth. After a suitable growth of the bacilli occurs, they are killed by heating in a current of steam, and boiled down to one-tenth of the original volume of the media. This is then filtered thru a Berkefeld or Chamberland filter, which separates the fluid from the bacilli. The filtrate contains only the soluble toxins and other byproducts of the bacilli. O. T. keeps well for a long period of time.

Later, Koch added bacilli to his tuberculin, and devised Tuberculin Residue and Bacillus Emulsion. Tuberculin Residue, T. R., or New Tuberculin, is prepared by grinding in a mortar 1 gm. of virulent tubercle bacilli taken from a six weeks old culture and dried in a vacuum. To this, 100 c. c. of sterile distilled water is added and the mixture centrifuged The supernatant fluid is poured off, and the process repeated several times. All fluids excepting the first are saved, until the total measures 100 c. c. after the addition of 20 per cent of glycerin. The glycerin acts as a preservative. Each cubic centimeter of this fluid should contain 10 milligrams of crushed bacilli.

Bacillus Emulsion (Bacillen Emulsion), B. E., is prepared from bacilli that have been filtered from glycerin broth media. The bacilli are ground, but not washed. One gram of the pulverized mass is added to 100 c. c. of distilled water and emulsified by vigorous shaking. To this, an equal quantity of glycerin is then added. Each cubic centimeter contains 5 milligrams of dried bacilli.

Bouillon Filtrate, B. F., of Deny, is a filtrate of glycerin alkalin bouillon broth on which tubercle bacilli have been grown. It is identical with old tuberculin, except that it is not heated. The mixture is said to contain the soluble toxins of the tubercle bacilli in addition to the culture media.

In 1901, Beraneck[2] made a tuberculin which he claimed was

less toxic and contained a higher percentage of specific substances. Its preparation is as follows: The filtrate of a culture of tubercle bacilli on nonpeptonized 5 per cent glycerin bouillon is heated, and the residue is mixed with 1 per cent orthophosphoric acid. To this mixture, an equal part of unheated filtrate is added. Beraneck thought that his tuberculin contained the desirable substances of O. T. and B. F.

Von Ruck's Tuberculin: The manufacturing of this preparation is more complicated than those that have been described. Cultures are reduced to one-tenth of their original volume by heating at 55° C. in a vacuum. This is a slow process and covers a period of several weeks. The mixture is then filtered successively thru filter paper and porcelain. An acid solution of sodic bismuth iodid is then added to the filtrate, and the fluid is filtered again and made neutral. Absolute alcohol is now added, and the mixture filtered. The precipitate is saved and washed with absolute alcohol. After drying, a 1 per cent aqueous mixture of the precipitate is made and filtered, after which it is ready for use.[3]

Dixon's[4] Tuberculin requires great care in preparation. It is essentially a mixture of equal parts of bovine and human tubercle bacilli that have been carefully removed from the culture media and thoroly dried. It is subjected to several washings of ether, which removes any remaining moisture and extracts the waxy capsule, and then it is thoroly ground. The ground bacilli are suspended in five parts of 8 per cent saline solution, thoroly shaken, and then allowed to stand for several days at room temperature. The mixture is then thoroly filtered and 0.5 per cent phenol added, then tested for sterility before using. One c. c. of the extract contains 0.2 mg. of the bacilli.

Bovine Old Tuberculin, B. O. T.: Rau and Spengler prepared an O. T. from bovine bacilli. It is made in the same manner as O. T., excepting that virulent bovine are used instead of human bacilli.

Polyvalent tuberculin: This is a polyvalent emulsion of tubercle bacilli. Polyvalent autotuberculin is an autogenous vaccine and may contain several strains of the organism. This form is rarely used at the present time.

Albumose free tuberculin, T. A. F., is tuberculin prepared on a medium that is free from foreign albumin.

Endotoxin, Tuberculin Purum, of Gabrilowitch, is tuber-

culin that is supposed to be free from all foreign proteins. It is prepared by precipitating the albumose with alcohol, ether, chloroform, and other chemicals, and washing them from the tuberculin after centrifugalization.

Partial antigens or partigens of Deyche-Much,[5] Leschke,[6] are antigens of the tubercle bacillus that are prepared by subjecting the bacilli to various diluted organic acids, i. e., lactic, malic, phosphoric, etc. The acids break down the bacilli into their component parts, which according to Much and Leschke are free fatty acids, a lipoid substance, a toxic principle, a neutral fat, a protein, various salts, and waxy alcohols. For a detailed description of the method of preparation the reader is referred to Much's book.[7] The various antigens may be used singly or in combination.

Friedmann's vaccine, socalled, is living human tubercle bacilli that have passed thru cold blooded animals, like the turtle.

While serum is not a tuberculin, it has been advocated as one of the means of biologic treatment of tuberculosis. Maragliano's serum is prepared by inoculating horses or cattle with tubercle bacilli of varying virulence, and the serum removed and treated similarly to that of diphtheria antitoxin.

Marmoreck's serum is similar to that of Maragliano's, prepared under slightly different conditions.

Other biologic preparations for the treatment of tuberculosis have been recommended from time to time, but the foregoing briefly describes the various types that have received greatest attention and have been most generally employed.

Of all the tuberculins, Old Tuberculin (O. T.) is most widely used. In ophthalmic practice, Old Tuberculin is frequently used for diagnostic purposes and Bacillus Emulsion (B. E.) for treatment. Some use O. T. for both. However, other forms are also employed.

Before discussing its use in diagnosis and treatment, let us briefly consider the theories that have been advanced to explain the action of tuberculins on the body. Koch believed that the reaction was due to the combined effect of the toxin and other byproducts of the bacilli in the tuberculin on the tubercle bacilli within the host. This was rather indefinite, and Kehler and Westphal suggested that the action of tuberculin, combined with that of the toxins generated by the bacilli in the host, produced the reaction. Marmoreck thought that

the tuberculin stimulated the tubercle bacilli in the body to secrete a fever producing toxin. Later, von Pirquet and Schick advanced a theory that is rather generally accepted. They suggested that the tubercle bacilli in the host stimulate the body to produce antibodies, or ferments, which, when in contact with tuberculin, produces a toxic substance that enters the blood stream and produces the local and constitutional reactions. Weil[8] believed that tubercle bacilli stimulate the body to produce amboceptors, which split the tuberculin into protein poisons, which cause a modified anaphylactic reaction.

Wolff-Eisner's[9] theory is most widely accepted. It is that tubercle bacilli in the human body stimulate two types of biologic reaction,—amboceptor or lysin formation for the purpose of breaking the tubercle bacillus down, so that it can be eliminated from the body and antibodies to counteract the toxic products of the bacilli. Lysins or amboceptors destroy tuberculin also. Healthy persons do not have specific amboceptors for tuberculin or bacilli, and when tuberculin is injected into the body no reaction occurs. However, persons with tuberculosis have varying quantities of amboceptors in the body, and after the administration of tuberculin, the amboceptors combine with it and produce a tuberculolysin, which is highly toxic and produces the reaction. If the tuberculolysin is entirely neutralized by the antibodies, no reaction occurs.

Kolmer,[10] following Wolff-Eisner's suggestion, states: "The general reaction may be explained as due to a general effect of the poison on body cells. The local reaction is caused by a concentration of the poison at the site of administration of the tuberculin, and the focal reaction is due to the fact that the cells about the lesion are more sensitive to the effect of the poisons that are supplied with a large number of senile or attached receptors (amboceptors) for the tuberculin."

Specificity.

It is upon the subject of the specificity of tuberculin that opinions differ widely. There is much discussion regarding this at the present time. The literature of the past is replete with discussions on this phase of the action of tuberculin. All agree that many tuberculous subjects react following the ad-

ministration of tuberculin. The question under discussion, however, is whether the reaction might not be produced with other substances to the same degree that it is with tuberculin. Thus, it has been demonstrated repeatedly that tuberculous subjects react following the administration of milk and other proteins.

This, like many other problems in immunology, is extremely difficult. It matters little from a therapeutic standpoint whether the reaction is specific or not. If improvement occurs following its intelligent administration, we are justified in advising its use. However, the advisability of using it as a diagnostic agent is quite another matter. I have always been inclined to attach great importance to a diagnostic focal reaction, and shall continue to think it of value unless further research proves it to be unreliable.

If proteins, other than those contained in tuberculin, produce focal reactions with regularity in tuberculous lesions, we cannot say definitely that the reaction is confined to tuberculous tissue. Other inflammatory areas might react in a similar manner. Further research is much needed on this subject.

We owe much to veterinarians for a large part of our present knowledge regarding the reaction of tuberculous subjects to tuberculin. In their hands, results can be confirmed immediately by autopsy findings. The work of Fraenkel,[11] Voges,[12] Kuhnan,[13] and others, has shown that in animals positive tuberculin tests were found in tuberculous beasts in 97 per cent of the thousands of autopsies that were done. They stress the fact that tuberculin is highly specific.

In adult human beings, positive reactions to tuberculin are frequent. When we consider that most adults show the presence of either active or healed tubercles (Nagele[14] and Burkhardt[15] found evidence of tuberculosis in 90 per cent of the adults whom they examined at autopsy), it is not surprising that positive general reactions are so frequent, especially so when we realize that minute lesions may produce hypersensitiveness. This fact alone should convince one that the general reaction is of little value when attempts are being made to diagnosticate the nature of an ocular lesion. As will be pointed out later, the focal reaction is the only one that can be relied upon when the lesion is in the eye.

It must be remembered that a positive general reaction indicates only that the individual is sensitive to tuberculin, and that a minute focus of tuberculosis probably exists in some part of the body.

Diagnostic Methods.

The ophthalmologist employs tuberculin tests to determine whether a given lesion in the eye is due to tuberculosis. It is not his object to discover whether the patient has active tuberculosis elsewhere in the body; that should be left to the internist, who is more competent in this field. For this reason, we should seek the assistance of the internist before launching out upon a course of tuberculin administrations for diagnostic or therapeutic purposes.

It should always be borne in mind that under certain conditions tuberculin is an exceedingly dangerous drug. If it is injudiciously administered in cases of active tuberculosis, it might so aggravate the disease that marked dissemination would occur or a fatal issue result. For this reason, the patient's general condition should be carefully investigated by one who is thoroly familiar with tuberculosis of the lungs and other parts of the body. If, after examination, the internist pronounces the patient free from clinical signs of tuberculosis, the ophthalmologist is justified in assuming the responsibility of the case and administering tuberculin. It is prudent, however, to have the patient examined from time to time, to be certain that no latent focus has been excited into renewed activity.

If active pulmonary, or tuberculosis elsewhere, is found, the ophthalmologist should require that the internist supervise the administration of the tuberculin. He should then satisfy himself with the observation and study of the ocular lesions. The determination of ocular reactions should be undertaken by the ophthalmologist.

I would not think of administering tuberculin for diagnostic or therapeutic purposes without first having the assurance of a competent internist that there were no signs of activity in the body, and that the administration of tuberculin was without danger. I am sorry that this is not the general practice.

The taking of temperatures, etc., before beginning a course of tuberculin should fall to the internist, and its consideration here shall not be discussed.

The various tuberculin tests are: Subcutaneous, cutaneous, conjunctival, percutaneous, and intracutaneous.

The subcutaneous test was first described by Koch.[1] It consists of injecting Old Tuberculin subcutaneously.

The cutaneous test of von Pirquet[16] consists in a local application of Old Tuberculin to a superficial abrasion in the skin. A positive reaction is manifested by redness, edema and induration of the skin. Three abrasions are usually made, two for the application of the tuberculin, and one to which no tuberculin is applied and is used as a control.

The conjunctival test of Wolff-Eisner[17] and Calmette.[18] This test consists of the instillation into the conjunctival sac of 1 drop of a 1 per cent solution of Old Tuberculin. A positive reaction is manifested by redness, chemosis, lacrimation and photophobia. This test is not without serious danger to the integrity of the eye.

The percutaneous test of Moro and Doganoff[19] consists in the application of an ointment of equal parts of lanolin and tuberculin around the nipple. A positive reaction manifests itself by the appearance of papules upon the skin in the region to which the ointment was applied.

The intracutaneous test was described by Mendel[20] and Mantoux.[21] They injected 0.05 c. c. of diluted Old Tuberculin into the superficial layers of the skin. They found redness and edema at the site of injection in positive cases.

The only test that is of value in ophthalmology is the subcutaneous. With the other forms, the reaction is local and indicates nothing excepting that the subject is sensitive to tuberculin.

The conjunctival test occasionally produces severe reactions, and its use should be discouraged. It is especially contraindicated in ocular tuberculosis.

The various reactions produced by the subcutaneous administration of tuberculin are local, constitutional and focal. The local reaction occurs at the site of the injection. It manifests itself by redness, swelling and pain. In severe cases, marked induration of the adjacent tissues, and occasionally necrosis, occurs. The constitutional reaction is indicated by headache, malaise, pain in the back, and fever. The focal reaction takes place at the site of the lesion. It is manifested by hyperemia, increased exudation and, in some cases, hemorrhage.

As has been previously stated, the focal reaction is the only reaction of value in diagnosticating ocular tuberculosis. If, after the injection of tuberculin, a lesion in the eye becomes definitely more hyperemic, increases in size, and an increase in the number of exudates or of the hemorrhages, occur, focal reaction has taken place.

Doses of 0.1 mg. of O. T. may be used for diagnostic purposes in lesions of the conjunctiva, cornea, sclera, iris, ciliary body, adnexa, and in some cases of tuberculosis of the optic nerve. Some prefer larger doses, and they may be used with caution. However, 0.1 mg. is usually a safe initial dose. In suspected tuberculosis of the retina in the macular region and of the retinal vessels, in addition to tuberculosis of the posterior segment of the choroid, the doses should be decidedly smaller. Some advise starting with from 0.000,005 to 0.000,025 mg.

Diagnostic tuberculin injections are usually given at intervals of forty-eight hours.

When dealing with the anterior segment, the doses are given as follows, 0.1, 0.5, 1.0, 2., and 5. mg., and occasionally 10 mg. Most observers are contented to exclude tuberculosis if no reaction occurs after 5 mg. have been injected.

For the danger zone cases, one must increase the tuberculin cautiously in the beginning, and if no reaction occurs, the doses may be rapidly increased in size. Individualizing and mature judgment are essential in these trying cases, and no definite rule of procedure can be stipulated with safety.

It is best to carry out tuberculin tests with the patient in the hospital, where he can be under constant observation and supervision, altho this is not absolutely essential, providing that the patient is intelligent and will cooperate. It is preferable to administer tuberculin in the morning, so that the temperature may be taken and recorded every four hours, and the eye examined at intervals of 8, 24 and 48 hours. If no general or focal reaction occurs after 48 hours, a second dose should be administered and the same procedure repeated. This should be continued until the maximum dose is finally reached.

The appearance of focal reactions differs somewhat with the location of the ocular lesion. In keratitis, the reaction manifests itself by increased cloudiness of the cornea, diminu-

tion in visual acuity, increase in the circumcorneal injection, lacrimation, photophobia and pain.

In scleritis, an increased congestion, induration and aggravation of subjective symptoms occur.

A reaction in iris tuberculosis would depend upon the type of iritis. In diffuse tuberculous iritis, the circumcorneal injection is aggravated, the aqueous becomes more cloudy and the precipitates on the posterior surface of the cornea increase in number, the iris becomes more swollen, the vessels, when visible, become more engorged, and the pupil contracts. In addition to this, the subjective symptoms become aggravated. In the nodular type, the greatest change is in the size of the nodule and in the injection of the blood vessels that approach it. Circumcorneal injection and the other accompanying symptoms become aggravated also.

The ciliary body reacts much the same as the iris. Visual acuity is often more disturbed than in cases of iritis, and secondary glaucoma is not an unusual accompaniment of the reaction.

Reactions in the retina and choroid are easily detected, provided the media are relatively clear. In the retina, reactions may be hazardous and result in marked loss of vision or extensive hemorrhage. The visual loss may be permanent, and the hemorrhages are frequently replaced by dense bands of scar tissue in the vitreous and retina. Haziness of the vitreous and increase in the number and size of exudates are frequently seen. Choroidal reactions are manifested by increased haziness of the vitreous, increase in the size of the tubercle, and occasionally by detachment of the retina, due to a sudden pouring out of exudate between the choroid and retina. Loss of visual acuity is also a prominent symptom.

Reactions in the optic nerve are manifested by increased haziness of the vitreous, increase in the size of the tubercle and an aggravation of the hyperemia of the disc. In addition to these symptoms the vision, if it has not been destroyed previously to the time of the diagnostic test, is diminished.

Old Tuberculin, O. T., is preferred by most ophthalmologists for diagnostic purposes. There is some difference of opinion, however, regarding the size of the dose and the interval of time that should elapse between injections. Probably, one of the reasons for the marked difference in opinion regarding the latter has been the failure to classify the cases and

regulate the dose to the individual. The dose of tuberculin should vary in accordance with the type of eye lesion. Doses that produce harmless reactions in the sclera would be fatal to the integrity of vision in tuberculosis of the retinal blood vessels. In certain types of retinal lesions, one should be content with a clinical diagnosis and not subject his patient to the danger of serious loss of vision.

The eye is an ideal location to study the effects of tuberculin on minute tuberculous foci, and the ophthalmologist is in a position to aid in either verifying or refuting the claims that have been made for tuberculin. By a systematic study and reporting of our findings in proven cases of tuberculosis, we should do much toward placing tuberculin on a scientific instead of a conjectural basis.

Therapeutic Use of Tuberculin.

We are more certain of the value of tuberculin in the treatment of ocular tuberculosis than we are of its diagnostic reliability. That it does good when properly administered in well selected cases, is beyond question. Whether other proteins might not do as well, is a subject that is now under discussion by students of tuberculosis. It has not been proved that its action is specific. The biologic and chemical nature of the tuberculins that are in common use leads one to believe that the reaction is possibly due to a selective protein phenomenon. It is said that Old Tuberculin contains the soluble toxins of the tubercle bacillus, but up to the present time, an exotoxin generated by the tubercle bacillus has never been demonstrated. It is difficult to explain its action excepting on the grounds of a foreign protein.

Bacillus Emulsion (Bacillen Emulsion), B. E., and Tuberculin Residue, T. R., on the other hand, contain ground tubercle bacilli, and on theoretic grounds might be capable of producing a specific immunologic reaction. Theoretically, these are the logical tuberculins to use for therapeutic purposes, and they are used for treatment more generally by ophthalmologists than are the other forms of tuberculin.

A tubercle is a localized reaction of the body to the presence of tubercle bacilli. It consists, microscopically, of a central collection of epithelioid cells which are phagocytes for the bacillus. In some tubercles, the bacilli destroy the tissues which they invade as well as the epithelioid cells, and produce

an area of caseation necrosis. Epithelioid and giant cells surround the caseated mass. The epithelioid cells are always surrounded by small round cells that consist chiefly of fibroblasts. The fibroblasts form scars. In a quiescent tuberculous focus, the lesion is entirely encapsulated by fibrous tissue which has developed from fibroblasts. When tuberculin is injected in sufficient quantities into the body of a tuberculous subject, one of the reactions is focal. If mild, the reaction is characterized by engorgement of the blood vessels which surround the lesion, which in turn stimulates the formation of fibroblasts and an increase in the number of small round cells. If the reactions are sufficient, scar tissue formation is stimulated and the lesion heals. However, if the reaction is severe, the formation of fibroblasts and epithelioidal phagocytes is retarded, the bacilli multiply more rapidly, and dissemination occurs. For this reason, it is imperative that a mild reaction in the region of the lesion be obtained before beneficial results can be expected.

It should always be our aim, in treating tuberculosis of the eye, to obtain an evanescent reaction at the site of the lesion. It is difficult to determine the size of the desirable dose. Here, again, individualizing is necessary. One should start with small doses and gradually raise them until the reacting point is discovered. The initial dose should be determined by the location of the tuberculous lesion. The same rules apply to treatment in this regard that apply to diagnosis.

It is the usual custom in Europe and in this country to begin the subcutaneous administration of B. E. and T. R. with 0.0001 mg. at from four to ten day intervals. Unless marked focal or constitutional reaction occurs, the dose is raised 0.0001 mg. at each injection period until 0.001 mg. is reached, after which time the increase is 0.001 mg. When a dose of 0.01 mg. is given, the additional quantity of 0.01 mg. is added to each dose. When 0.1 mg. is given, the intervals are prolonged to two weeks and the dose raised 0.1 mg. with each injection, until it is 1.0 mg. After this, the time between administrations should be prolonged to intervals of two or three months, and continued at this rate for a period of a year or more. When the larger doses are being given, one should be careful to avoid injecting into areas that have received doses previously, because of the danger of severe localized reactions.

This method of administration is comparatively safe in all forms of ocular tuberculosis excepting those which occur in the retinal vessels, the macular region of the retina and the choroid. Here, as in its diagnostic use, tuberculin must be given with extreme caution to prevent reactions that may result in serious permanent damage to vision. When this so-called danger zone is involved, a safe initial dose is 0.000005 or 0.00001 mg. I have seen serious hemorrhages following the subcutaneous administration of 0.0001 mg. of B. E. in cases of tuberculous retinal periphlebitis. The increase should be according to the principle in the method described above, i. e., increasing 0.000005 mg. until 0.00001 is reached, then adding 0.00001 mg. until 0.0001 is given, etc.

If at any step during the administration of the tuberculin a severe reaction should occur, the dose should either be lowered to one that was known to be below the reacting point, or the treatment discontinued for a short time and resumed at a smaller dose. Individualizing is essential in every patient, and a set rule can not be laid down for all cases. The foregoing method, however, has been most satisfactory in the average case, and in the hands of competent observers.

It must always be born in mind that mild reactions are desirable, and that when severe, they jeopardize not only the eye but the body in general.

I have had no experience with tuberculin other than with O. T., B. E. and T. R. I have not undertaken the use of the other forms, because the majority of observers are agreed that some are worthless and the others have no merits not possessed by one of the foregoing.

The fact that tuberculin alone will not cure tuberculosis of the eye does not seem to be generally appreciated by ophthalmologists. I have heard tuberculin condemned as useless many times by men who expected it to accomplish a cure or marked improvement after five or six injections. Tuberculin is not a specific in the same sense as diphtheria antitoxin. It is only an aid, and until it is considered as such, its action will never be understood. Hygienic, local, symptomatic and other forms of treatment are just as essential in the management of a case of tuberculosis of the eye.

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TUBERCULOSIS OF THE MIDDLE EAR AND MASTOID.

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The frequency of middle ear suppuration in patients with pulmonary tuberculosis was realized as early as the middle of the last century by Toynbee[1] and von Troltsch.[2] Wilde[3] had noticed the painless onset of tuberculous otitis media, followed by discharge, and the peculiar appearance of the membrana tympani, thirty years before the discovery of the tubercle bacillus by Koch in 1882. Eschle[4] found tubercle bacilli in the discharge from the middle ear in 1883, and Habermann[5] first demonstrated giant cells and tubercle bacilli in the tympanic mucous membrane.

Politzer,[6] in 1865, described the characteristics of tuberculous otitis media. Politzer[7] later made a histologic examination of the middle ear, and described his findings in 1882. Schwartze[8] described in 1878 small tubercles in the mucous membrane. However, the disease does not receive the serious consideration which it deserves, as a study of the literature of the subject shows, and the diagnosis, in the milder types, is likely to be overlooked. The etiology of all cases of suppurative otitis media should be determined whenever possible.

Tuberculosis of the middle ear is more frequent than is generally supposed, because it is easily overlooked, even in patients suffering from pulmonary tuberculosis. This is attributable to two things: First, the absence of pain which usually forces the patient to seek the otologist's advice; and second, when mixed infection is present, the real cause, viz., the tubercle bacillus, is not easily obtained from the pus.

Mathews[9] says, "There are numerous considerations which combine to render tuberculosis of the middle ear and temporal bone a subject of great interest and importance, both clinically and pathologically. The number and variety of the channels thru which infection may take place, the frequency of the condition, its occurrence in infants, the gravity of the condition

and of its possible complications, the multiplicity and diversity of its signs and symptoms, the urgent necessity for drastic treatment, the possibility of absolute diagnosis and ultimate cure are the most important of these considerations."

The history is often quite typical, because the patient will tell you that the first thing he noticed was a feeling of moisture in the external auditory meatus, and that, when he placed his finger in the canal, he encountered pus. Mathews says, "The absence of pain is not peculiar to this condition, but is found in tuberculous osteomyelitis of any bone prior to the involvement of surrounding structures or the advent of mixed infection." Tuberculous lesions are generally free from pain, and tuberculous otitis media is no exception.

Mathews[9] furthermore says, "The occurrence of discharge from the ear is not infrequently—in the absence of pain—the earliest symptom noticed. This discharge presents many points, apart from its mere chronicity and the absence of associated pain, which may justly raise suspicion that the case is tuberculous."

An additional history of pulmonary tuberculosis serves to put the otologist on his guard. Tuberculosis of the middle ear can and does occur, of course, in patients who have not had pulmonary tuberculosis. The complete absence of pain preceding the discharge is a very important point in the history. If pain is present, this is nearly always caused by mixed infection. The number of pus producing organisms naturally determines the amount of pain. If there is severe pain preceding the discharge of pus, the tuberculous etiology can the more easily be overlooked, unless a careful bacteriologic examination is made. Robinson's[10] patient had tinnitus before the drum ruptured, but no pain. The absence of tubercle bacilli at the first examination counts for very little. Personally, I have not obtained a history of pain if the otitis media was caused entirely by tubercle bacilli. Leegaard[11] believes pain is nearly always present preceding the discharge, but perhaps his patients had a mixed infection.

Briggs[12] mentions the possibility of an ear infection originating thru the lymph channels from a brain tubercle. Graham,[13] in commenting upon this, says, "It is interesting to note that Habermann[14] has since described such a case." However, extension from the brain must be rare indeed. Infection, especially in infants, but in adults as well, must fre-

quently reach the middle ear and mastoid thru the Eustachian tube. At least, in adults suffering from pulmonary tuberculosis and with sputum laden with tubercle bacilli, this mode of infection is likely the most frequent. The tube is shorter, more nearly horizontal and more patent in infants than in adults. Metastasis thru either the lymph or blood stream must be important. In infants and young children, tuberculosis of the lymph glands is frequent as compared to pulmonary tuberculosis. Therefore, infection may take place thru the lymph channels. Infection from the external auditory meatus, when a perforation has previously existed from other causes, is quite possible. Emphasis has often been placed upon the fact that the tonsils and adenoids become infected first, with secondary foci in the temporal bone. Other avenues of infection may, in occasional cases, be important, but time will not permit of a free discussion of all of these at this meeting.

Examination of the affected ear often reveals a small amount of thin seropus along the floor of the canal, in some instances obscuring a good view of the drum. After the seropus has been wiped away, multiple perforations are usually found in the membrana tympani. These are often very small perforations, but they may gradually increase in size during the subsequent months. If several coalesce to form one larger perforation, or even two or three larger perforations, the picture becomes less and less typical. Granulation tissue about the margins of the perforations is more frequently encountered than in nontuberculous suppurative otitis media. Gaudier[15] regards the presence of profuse granulations as a suspicious sign. However, there are many exceptions to this. The granulation tissue is paler, and consequently less vascular, than when caused by other than tubercle bacilli. The tendency of staphylococci to invade the middle ear, from the canal, in any patient with suppurative otitis media of a few weeks' duration, is well known.

The mastoid is rarely tender in the early stage of the otitis media, and may not be invaded by the tuberculous process beyond the antrum for months or even years. This is especially true in patients suffering from pulmonary tuberculosis. In fact, the mildly insidious onset is one of the chief characteristics of the disease in adults. Patients with incipient pulmonary tuberculosis often have enough immunity to prevent an acute onset. It is not uncommon to see the disease remain

confined almost entirely to the middle ear, in adults, for years. Rough bone is not found upon examination with a probe; the ossicles are not necrotic; there is very little discharge and very little granulation tissue. The roentgenograms of the mastoid show, from time to time, only a slight cloudiness, and the hearing is quite good.

In contrast with these mild types, are those of greater severity in patients with advanced pulmonary tuberculosis and consequently poor resistance. Mixed infection also favors a severer type of otitis media. The perforation is larger, there is more granulation tissue, more pus; there is rough bone; there are necrotic ossicles, more loss of hearing and a more rapid invasion of the mastoid. Roentgenograms of the mastoid often show second and third stage mastoiditis.

The severest types of tuberculosis of the middle ear and mastoid are usually encountered more frequently in infants than in adults, and the cervical glands are also tuberculous. The disease spreads rapidly from the middle ear to the mastoid, and often involves the whole temporal bone. The membrana tympani and ossicles are rapidly destroyed, so that pieces of the ossicles are found in the pus. Sequestra of bone have to be removed surgically from time to time, and the cervical lymph glands also require removal. The temperature is often over 100° F. Paralysis of the facial nerve occurs in approximately 50 per cent of the patients, and the labyrinth, including both the static labyrinth and cochlea, is often destroyed. Meningitis is a frequent terminal complication.

Graham[13] has classified his cases as follows: "Lupoid, infiltrating, fungoid, necrotic, myringitis diffusa of Möller,[16] and fibrinoid." His classification is based upon the clinical pathology, found either at the time of operation or at the autopsy, and has much in its favor.

During the first few weeks of the disease, the bacteriologic examination of the seropus may reveal tubercle bacilli. However, repeated examinations by a competent bacteriologist may fail to find even a few tubercle bacilli. Failure to find tubercle bacilli in the discharge counts for very little. In fact, it has the same significance as failure to find the bacilli in sputum, but a positive finding is of great value. Staphylococci are easily found, but are misleading. They represent a secondary infection from the canal. The tubercle bacilli are prone to re-

main in the tissues of the middle ear, hence the difficulty of finding them in the discharge.

In 1911, I brought back from Berlin a small glass aspirator, devised by Dr. Max Halle, which is very useful for aspirating pus from the middle ear. The aspirator should be sterilized by boiling, and the canal thoroly wiped out and cleansed with alcohol. This aspirator fits tightly into the canal, and with the aid of the hand bulb, a few drops of seropus can sometimes be obtained from the middle ear. If staining methods fail to show tubercle bacilli, a guinea pig experiment usually does.

Predisposing causes, either immediate or remote, are important factors. Immediate predisposing causes are as follows: Syphilis, either congenital or acquired; tuberculosis in other organs of the body, e. g. pulmonary tuberculosis in adults, or tuberculosis of lymph glands or of bone in infants and young children; measles, scarlet fever, whooping cough, more rarely the other contagious diseases of childhood; sinus diseases; rickets; poor food; overcrowding; unhygienic environment; loss of sleep; lack of fresh air and sunshine, etc. Remote predisposing causes are as follows: Living with tuberculous people; nasal obstruction; diseased tonsils and adenoids; improper clothing; repeated colds, etc.

The examination made by the otologist is by no means complete without a thoro general physical examination by a competent internist, roentgenograms of the chest and mastoids, urinalysis, sputum examination, serologic tests, etc. In patients suffering from pulmonary tuberculosis, the primary focus is usually in the lungs; but in the patients with normal lungs, the primary focus is often in tuberculous glands. This is especially true in children, because pulmonary tuberculosis is rare in children, while tuberculosis of the lymph glands is common.

When Dr. I. S. Fraser of Edinburgh was an honor guest at Atlantic City, New Jersey, of the American Otological Society, the American Laryngological Association and the American Laryngological, Rhinological and Otological Society, in 1923, I heard him make the statement that tuberculosis of the middle ear is frequently found in the children of Edinburgh, Scotland, because the Scotch dairy herds are not tuberculin tested, with the result that the milk often is infected with bovine bacilli. He showed many interesting slides of tuberculosis of the middle ear, mastoid and labyrinth. Ravenel[17] says, "Laboratory

investigations have shown beyond question, that bovine tuberculosis is transmissible to human beings, and that the lesions produced do not differ in any way from those caused by human bacilli."

Otitis media is nearly always much more extensive in children than in adults. The mastoid or even the entire temporal bone may be involved. Long[18] has reported tuberculosis of the middle ear and mastoid in a baby of 11 months, cured by a radical mastoid operation. Graham[13] has reported tuberculosis of the middle ear and cervical lymph glands in an infant, with a fatal termination from a general tuberculosis, at 72 days of age. Large sequestra of bone may require removal surgically from time to time, over a period of several years. The cervical glands are usually involved, and may also require removal surgically. Crockett[19] has emphasized the swelling of the preauricular lymph gland. He says the cervical glands are less likely to recur, following their removal, if the middle ear and mastoid have been operated upon first in the case of small children. He advocates repeated operations, if necessary, to remove the tuberculous foci from the temporal bone.

Horne[20] believes the early glandular involvement is the result of primary tuberculous middle ear disease, and is rarely found in secondary involvement.

Leegaard[11] states that tuberculosis of the middle ear and mastoid is most frequently found in children from 1 to 10 years of age. He believes paralysis of the facial nerve occurs in from 19% to 65% of the cases. Milligan[21] found facial paralysis in 45% of his series of tuberculous cases, and in only 2% of his nontuberculous cases. However, Lake[22] encountered facial paralysis in 4 of 658 consecutive cases of suppurative otitis media. Grunert[23] believes facial paralysis is almost pathognomonic of tuberculosis of the middle ear and mastoid.

Levy[24] says, "The vast majority of suppurative processes in the middle ear occurring in tuberculous patients do not go on to destruction of bone; therefore, surgical measures have but limited usefulness." This corresponds, in a general way, with the experience of all of the otolaryngologists in Colorado, so far as I know, and at least with my own experience, except with children. In infants the disease is likely to be more serious.

Milligan[21] advises operation when the extent of the disease is not too great, and the general condition of the patient not too hopeless.

The diagnosis is based upon the insidious onset; the absence of pain before the membrana tympani ruptures, except in patients with a mixed infection; multiple perforations in the drum; abundant pale granulations, and last, but not least, the finding of tubercle bacilli in the pus. The general physical examination, laboratory tests, roentgenograms of the chest and mastoid, are all important aids in diagnosis. Later in the course of the disease, fever, necrotic bone, bony sequestra, deafness, facial paralysis, etc., are only more positive evidences of the seriousness of the disease.

The prognosis in the mild type is usually very favorable, but is serious, at best, in the severe type. It is often wisest to keep such patients under observation for some time, before making any very positive statements about the outcome. A guarded prognosis is better than hasty judgment.

The treatment decided upon as most suitable must of necessity be determined by the stage of the disease, the severity of the disease, the patient's general condition of health and age. The mild type requires daily cleansing of the external auditory meatus, as often as the pus collects, followed by instillations of 10 per cent phenol in glycerin, 2 per cent mercurochrome in water, 4 per cent protargol in water, etc. The patient's general health should receive every care and attention from an expert internist, preferably one skilled in tuberculosis.

Heliotherapy is of great advantage, and can often be applied to the middle ear more easily than to the larynx. A large aural speculum, with as large an opening at the small end as can be comfortably maintained in the external auditory meatus, is best. The patient should secure an aural speculum, or should be shown how to make one from rather heavy and stiff, white, glazed paper.

The membrana tympani and middle ear should be exposed to the sun's rays, in the open air, for two or three minutes daily the first week. The exposures should be increased a few minutes each succeeding week, until each daily treatment consumes from ten to twenty minutes. The ear of an adult should be pulled up and back, and the head of the patient, with the speculum in place, held so that the sun's rays can strike the drum and middle ear. The external auditory canal should always be cleansed immediately before each treatment.

Rest, fresh air, sunshine for the entire body, plenty of good food, climate, etc., are all important for the treatment of tuberculosis of the middle ear and mastoid, just as they are for tuberculosis in other organs of the body.

The severer types often require a mastoid operation, provided the patient's general health will permit. If the condition of the mastoid is not urgent, the operation often has to be put off until such time as the general health will permit. Crockett[19] favors surgical removal of sequestra of bone and tuberculous cervical lymph glands in infants as often as these form, over a period of several years. The severest cases require experience, mature judgment and surgical skill if a good result is to be obtained.

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DISCUSSION OF SYMPOSIUM ON TUBERCULOSIS.

DR. ROBERT LEVY, Denver: I was rather amused to find that Doctor Spencer had resurrected a paper of mine, read in 1903 before this organization in Indianapolis—amused because I read the paper myself a day or two ago, and found there were some features about it that would not hold water today, I am sure. However, I believe the statements Dr. Spencer quoted may still be true, namely, that we see comparatively few cases in which bone is involved in a necrotic process in middle ear tuberculosis. That statement also requires some reservations, for the diagnosis of middle ear tuberculosis is not easy. There is no reason why a tuberculous individual should not have a chronic suppurative otitis media from other causes, and many authorities have called attention to the fact that many of the symptoms of tuberculosis, many of the complications of suppurative processes in the middle ear in tuberculous individuals, are due not to tuberculosis, but to a mixed infection.

As to the incidence of tuberculosis in the middle ear, I think it is generally agreed that the disease is much more frequently seen in children than in adults, and also that it occurs more frequently in very young children—in tuberculous infants. This is explained on the ground that tuberculosis of infants is a milk borne disease, the bovine tubercle bacilli finding access to the economy, so that in very young children, milk fed children especially, tuberculosis may develop either by extension thru the Eustachian tube or thru the blood and lymph currents.

The symptoms leading to the diagnosis of tuberculosis of the middle ear have interested me, in that the very earliest manifestation is not always a discharge. We have been able to make a diagnosis before the discharge by recognizing certain prodromal manifestations, namely, a sense of fullness, slight deafness and some tinnitus in an individual suffering from tuberculosis. In these cases, very little change can be seen in the drum membrane, except possibly a certain cloudiness and a loss of the usual landmarks. The problem here is incision at this particular time. I think, generally speaking, that in individuals beginning with such symptoms as I have enumerated, it is wiser to incise the drumhead, even before the discharge. We have nothing to lose and perhaps something to gain. Of course, perforation will result, if it is a definite tuberculous invasion, if we do not incise, the perforation being due to necrosis from deposit of tubercles. Therefore, if there is definite infection of the middle ear in a tuberculous individual, even before discharge the incision should at least be considered seriously.

One word more about diagnosis. We have always been led to believe that a perforation coming on without pain, preceded by a moderate degree of discharge of a thin, serous, mucoid character, is indicative of tuberculosis, especially when found in a tuberculous individual. Generally speaking, I think that is true. The typical diagnostic points are the lack of pain, the multiplicity of perforations, and their size.

The finding of the tubercle bacillus cannot be depended upon. Other acid fast bacilli may be found in the ear. I think that is one of the reasons for erroneous diagnosis—the difficulty of distinguishing between tubercle bacilli and other acid fast bacilli.

DR. THOMAS J. GALLAGHER, Denver: I am very much interested in the preperforative stage of tuberculosis of the middle ear. Doctor Levy

has expressed some doubt as to the desirability of incising the ear drum. I am decidedly in favor of incising the membrane as soon as a diagnosis of tuberculosis is made. For many years, it has been our custom to make two incisions in the membrane, one anterior to the malleus, and the other posterior. These are two linear incisions, extending from the anterior and posterior folds of Shrapnell's membrane directly down to the annulus tympanicus. I am not in favor of the ordinary single curvilinear incision made posteriorly only. Our idea is to preserve a strip of membrane holding the malleus within it, which increases the tension of the ossicular chain. If we succeed in limiting the destruction to the ear drum, which we very often do, the hearing will be better preserved. The rest of the treatment should be carried out in accordance with modern methods.

The use of suction is very valuable. Two methods of suction may be used, one in which the canal is completely closed, and the other in which a cannula is used thru the perforation, cleaning the different parts of the middle ear. Doctor Carpenter of this Society has used suction not only for the cleansing of the ear, but has succeeded in drawing the secretion from the mastoid process, as we also have done. We use a very simple hand apparatus for the suction. When the external canal is completely closed with the suction apparatus, if the Eustachian tube is unduly patent, we close the tube in the nasopharynx by using a curved applicator, otherwise the secretions may be drawn up from the nasopharynx.

The question of operating upon the mastoid itself is to be decided by each operator, taking into account the age and condition of the patient. The anesthetic should be carefully selected. Rest in laryngeal tuberculosis we have all found very beneficial, but I am strongly in favor of instituting active interference by destroying the tubercle by means of the galvanocautery. This is followed by our usual formaldehyd treatment, which I advised in an article read before the American Medical Association, 1898, in Denver. The direct inspection of the larynx, using the instruments which we now have, has simplified matters very much. The use of heliotherapy or the quartz lamp is, of course, very beneficial. The tuberculous glands should not be surgically interfered with until irradiation has been thoroly tried.

Dr. W. Likley Simpson, Memphis, Tenn.: This summer, while visiting in Vienna, I had the opportunity of seeing and making a study of a light that is now being used exclusively in the Hajek clinic for the treatment of tuberculosis of the larynx. This light has been worked out by Wesley, an assistant in the clinic. It is a powerful arc light, having about 2,000 candle power, and it is used either by the direct method or by means of mirrors. Hajek now has in that clinic about 100 cases, which were rather advanced cases of tuberculosis of the larynx, that have been pronounced cured. These cases were watched over a period of several months. It is rather an unusual light to me. They are making no special claims for it, but as you know, in this clinic, they formerly used the galvanocautery almost exclusively, but now they use this light with very much better results.

Dr. Walter R. Parker, Detroit: I wish first to say that I agree in every respect with what Dr. Finnoff has presented in his paper. I would feel much at a loss in treating certain chronic inflammatory conditions of the eye without feeling that we had as a resource the occasional use of

tuberculin. I have followed the mode of administration as advised by the author. I cannot see how anyone can say that good results are not obtained from the use of tuberculin. It may not be a specific; perhaps the benefit comes from the use of a foreign protein.

The caution Dr. Finnoff gives in regard to the therapeutic tests is certainly well taken. These patients should not be given tuberculin until after they have had a general examination, and pulmonary or general tuberculosis eliminated. But in certain cases of scleritis, uveitis and some inflammatory conditions of the fundus, nothing takes the place, in my mind, of the use of tuberculin.

Dr. H. B. Young, Burlington, Iowa: I wish to say a word in connection with the internist's presentation of the subject. I feel that I have a peculiar advantage in that respect. For eight years, I fought a losing fight with tuberculosis in the person of my daughter. She had all the modern methods of treatment—heliotherapy, pneumothorax, etc., and she was twice pronounced in an arrested condition. It is easy to prescribe rest in the treatment of tuberculosis, and she had bodily rest in plenty, but if we do not have contentment of mind, it makes no difference what the treatment is. Rest in the treatment of tuberculosis is often passed over as an easy thing to do, but I want to emphasize the idea, that while bodily rest is important, we must also have contentment of mind.

Dr. Wm. H. Crisp, Denver: I simply wish to suggest, that in the treatment of ocular tuberculosis, we do not seem to hear as much about bodily rest as we do in other forms of this disease. It seems to me that that is a point we should not overlook. We may try tuberculin, and in some cases we may think we have good results, but there is usually a very problematic situation as to just how far tuberculin is doing anything for the patient. Now and then we have the experience of a patient refusing to be put under tuberculin treatment. Of course, the reaction is to decline to take care of the patient, but I think that is extreme. I had one or two cases in which I had such refusal, and in which I had demanded, even altho the patient appeared to be in good physical condition, a very large amount of rest in bed—perhaps not the whole day, but a certain proportion of the day being spent in bed, with short intervals of activity, and I feel that this was a very important factor in producing arrest of the ocular condition.

Dr. Frank R. Spencer, Boulder, Colo. (closing): I appreciate the discussion of the paper, and heartily agree with all that has been said. I think Dr. Gallaher's mention of early myringectomy is desirable. The unfortunate thing is that the otologist rarely sees these patients until they have a discharging ear. They have so few symptoms and they do not often seek relief early.

I think suction is desirable,—I have used it some. But I cannot quite agree with Dr. Gallaher that you can aspirate mucopus thru the Eustachian tube. I feel satisfied if I can aspirate pus from the middle ear and turn it over to the pathologist for examination.

Dr. William C. Finnoff, Denver (closing): This paper has dealt entirely with tuberculin in ophthalmology. I have not discussed the treatment of ocular tuberculosis. That could not be covered in the period of time allotted to the paper, so I have not mentioned rest and the other methods of treatment that are absolutely essential.

THE OPHTHALMOLOGIST AND OTO-LARYNGOLOGIST IN PERIODIC HEALTH EXAMINATIONS.

E. H. Cary, M.D.

DALLAS, TEXAS.

In recent years, publicity thru many agencies other than our profession, has created an ever increasing demand for periodic health examinations. The profession, as a whole, cannot be credited with having brought to its present status what may prove to be a most useful service to humanity, largely because there is no health standard which can be pointed to as acceptable, and there is confusion in the mind of the average medical man.

Primarily, the medical schools have not been interested in the subject. The fundamental teaching, didatic and clinical, revolves around morbid anatomy. Hospital interne service, after graduation, is focused upon the sick, and there is no interest in the normal man, hence the relative value of focal infections here and there appearing in what might be considered a well man makes no appeal. Focal infections are not classified and evaluated with the view that such correlated data could be used for suggesting help to the apparently normal man.

There can be no definite health standard that is satisfactory to the patient, which does not take into consideration, not only the man free from symptoms of disease, with negative laboratory findings, but to my mind a correct analysis of all the focal points of infection which are usual, all of which should be properly explained to the patient. With this interpretation, the responsibility of an examination becomes more formidable than the average conscientious practitioner is willing to assume by himself.

Educating the public to have periodic health examinations, suggests it is our duty to see that the general practitioner realizes there can be no satisfactory health standard which loses sight of the relative value of focal points of infection, which have not yet produced symptoms which he would easily

recognize. A discriminating public often becomes confused and disappointed when necessarily examined in different places, by different doctors, all of whom may have had excellent medical training, but who vary in technic, interpretation and technical expression, much of which can be harmonized, hastening a better understanding of what we mean when we speak of health standards and normal men.

I fully appreciate the fact that we who limit our work are not in accord at all points. Our interpretations of a given condition stated offhand, when not in consultation, could easily be distorted into a wide difference of opinion, hence before saying too much to the general practitioner, it would seem that we could profitably study relative values of prognosis in these potentially sick individuals who are without symptoms of disease.

First impression suggests that it is impossible to standardize thinking on the part of the Doctor, or possibilities of an individual's resistance to infections either concurrent or dormant. However, if tabulated experience, based upon the best of training, means something to the individual doctor, why not make a serious effort to standardize this accumulated knowledge of many doctors, to the extent that medical men can reasonably use it in more accurately estimating probabilities when confronted with the task of periodic health examinations of the supposedly sick or well man?

Herein lies much of the charm in the practice of medicine. Every patient presents a classifiable problem, but usually discriminating judgment is most important as to classification, since cause and effect, are at times, difficult to separate, and good observers may feel justified in their views, even if, seemingly, at variance. I am inclined to think the vast majority of the older practitioners will never become interested in these examinations to the point where they will not say to their patients in families where they have practiced for many, many years: "John, you are all right. I know you like a book, and I would not trouble if I were you about all this modern propaganda, which is in the interest of these young specialists." This type of practitioner must be educated primarily in the technic of a general examination, and that he can receive valuable assistance from us; then he will see the wisdom of it all. So we must consider this question and make our knowledge available. The value of an examina-

tion will be increased as intelligent cooperation is manifested, hence we must have definite ideas concerning the relative values of infections in remote places, which frequently harbor germ life for many years without any apparent loss to the physicial economy of its host.

We have all seen many incurable conditions which may have been the result of tardy diagnosis, such as we see in cardiovascular diseases, pernicious anemia, arthritis deformans, etc., all of which diseases are but the result of accumulative poisons. An early analytic study of the individual, with the view of the elimination of all such probabilities, might prevent numbers of persons we see becoming hopelessly impaired. If our present conception of disease is correct, then an early recognition of infection and a better understanding of its incipient devastating possibilities, would prevent much of what we see as sequelae, and the individual might avoid that unhappy physical state with reduced resistance where any concurrent infection becomes formidable.

With an outlook of this kind, the Biblical three score years and ten would seem too short. Modern speculative scientific statisticians have for a long time believed the longevity of the individual whose existence was lived according to known scientific principles, could easily exceed the old accepted allotted time.

No one who has seen the old grape vine, now 158 years old, at Hampton Court, near London, perfect in its growth and fruit, will fail to recognize that its longevity is the result of the proper relation it has to its food supply, its atmospheric surrounding and the care men have given to its body, keeping from it those poisons and parasites which would have long since destroyed this beautiful productive natural plant, which has the quality of life or death, but has the unusual distinction of care scientifically applied, which will be carried on indefinitely, the result being a wonderful demonstration of what is possible.

Recently, the distinguished Indian Savant, Sir Jagdis Chandra Bose, at the Oxford conclave of Scientists, demonstrated that plants have systolic circulation (heart action), and that it can be stimulated or depressed, as in animals, reminding us that we are only a part of the whole. Being subject to the same law, frequently responsive to the same poisons, we but vary in resistance and susceptibility, in inherent strength and weakness; hence the individual becomes a

complex problem, making it impossible to have an inflexible standard to guide physicians, unless they have the broadest conception of the whole question, intermingled with scientific training and a wholesome cooperative sense.

To determine a correct health standard for the individual will always be a problem of the medical practitioner. He must have in mind general conclusions, which have been the result of a study of the mass, such as current views upon heredity, sequelae of early diseases, effect of weight, mental attitude, habits, focal points of infection, with susceptibilities, strains from overwork, including eyestrain, environments, meteorologic and sociologic, all of which influence the individual and require interpretation. Having this data in hand, one can then see the opportunity the real doctor has to demonstrate his ability.

To claim that all focal points of infection likely to lower the established index of health fall within the scope of the rhinootolaryngologists, is not my purpose, and it would be ridiculous so to construe anything I might say on the subject, but to deny that a vast number of interesting physical phenomena are the result of these infections, either directly or indirectly, is to shut one's eyes to the obvious. So many diseases seem to be the culmination of these underlying conditions, a vast number of respiratory susceptibilities would never occur if the individual were not a carrier of germ life, and when circumstances conspire, they break thru the weakened barrier. I have already intimated that cardiovascular, and many of the chronic diseases, are ultimately the result of cumulative poisons for a long time tolerated without symptomatology, which suggest that a searching investigation of the parts under our care is most essential, not only to determine the present health status of the individual, but future possibilities. We should provide that information which would aid in standardizing the statements of the practitioner to the patient. Incomplete statements bring ridicule to the profession, when a general practitioner has essayed to pass judgment without taking into consideration specialized knowledge.

As to whose judgment, that of the general practitioner or the specialist, is soundest in evaluating the possibilities of focal points of infection, depends upon the man, granting the basic knowledge is very much the same. The question of

prejudice, recorded observations, the question of being able to observe, the question of having an analytic mind, even the question of being interested in the following up of one's observations, have to be considered before one would offhand grant that a general practitioner's experience is in any way better than a specialist, or that he is comparable in properly evaluating focal points of infection.

Ophthalmologists, as well as the rhinootolaryngologists, very early deal with symptoms of disease, when a general observer would classify the patient as well. The ophthalmologist who understands the use of his perimeter, and who consistently charts every case, has an indication which leads to questions involving the pituitary body, as well as other parts of the brain. When the ophthalmoscope is used understandingly, there are many positive and negative conditions of the nervous system unfolded for the interpretative gaze of the wise medical man, whether he be the general practitioner or confines his work to the limited field of ophthalmology.

Headache or pain, in and around the eye, is usually conceded for analysis to the ophthalmologist. His differential diagnosis is most important. Many patients are permanently relieved of this symptom—some temporarily. The ophthalmologist should be able to find the associated cause quickly; this may lead him to use the rhinologist, endocrinologist, neurologist or internist, or he may be used by them, for he is an important link in the analytic chain surrounding humanity for its good.

As an ophthalmologist, you can see a typical case of so-called albuminuric retinitis occurring in a patient where you are unable to discover vascular changes elsewhere in the body. You can see this when reduction of vision is the only symptom, this occurring before the kidney glomeruli have become irritated. The ophthalmologist has the opportunity of pointing the way to the source of infection, and will be able to demonstrate the very exudates from whence the toxins spread.

In time, of course, the changes in the kidney and other organs would make a diagnosis very simple, but the point I wish to make is, that the general practitioners can depend upon us to help in early recognizing conditions which save the individuals from lesions, which if not seen early, become often too far advanced for nature to repair.

As to the rhinootolaryngologist, indications for his aid, within his field, are multiplied by the number of recesses

which harbor infection. As simple as it appears, there is a mechanical basis leading to many troubles which comes early under the scope of his observations, and many of the infections later necessitating care could be avoided if mechanical problems, for instance nasal obstructions, were relieved when first seen, for individuals have potential possibilities for sinus infection, which should not remain with their accumulative underlying qualities.

We would say to the medical men who treat other parts of the body, that it requires a most careful analysis and the most searching investigation before one can in truth eliminate the sinuses as a source of infection in a given case. It requires X-ray, transilluminations, washings, and the most careful analysis of the symptomatology and the history of the patient before a man of experience will dare say that there is not absorption from the field under his observation.

I have raised the question: "When we are properly trained and observant, do we not interpret laboratory findings as intelligently as any other group?" My answer is "yes." Assuming that we have had hospital experience, both general and special, where the best known all inclusive routine prevailed, having absorbed the philosophy of this with due appreciation of its value, this routine being one of the big things the young doctor gets out of hospital training, then with this attitude of mind, it is a matter of practice applying daily to each case the acid test of general and specific knowledge. If this is done, your special knowledge becomes a supplement to general training, developing skill in diagnosis as well as ability to interpret the things we have been talking about, infections and laboratory findings. With this in mind, I think we have the advantage, certainly possessing information greatly needed. When the specialist fails to measure up to this conception, or where he has lost interest in general medicine, and has evaded contact thru association in its practice, to that extent he must lower his flag and accept the opinion of others.

In the practice of medicine, one may have the art, by specializing can become an artist, or even descend to be an artisan. In the accumulation of special knowledge, neglecting general basic knowledge, true art cannot be achieved; lacking appreciation of the greatness of art, one becomes an artisan, for an artisan has lapsed into a mechanical attitude, automatically accomplishing a daily task, forgetting to be the artist. One

must not permit oneself to be isolated and limited to any part without due regard to the whole.

To obtrude the statutory specialist into this discussion is difficult, inasmuch as it changes the current of thought, but these gentlemen on the periphery of medical practice abound in our land.

When periodic health examinations become popular, there will be a marked increase in reference work on the part of the general practitioner. With this in mind, is it good for the public that the qualified specialist in our field should remain inactive in the education of the general practitioner to the equality of service required? I fear we assume the medical men appreciate the difference; recent graduates may, but the older practitioners frequently seem unable to differentiate an oculist from an optometrist.

We recognize two groups within the rank of the optometrist. One group, apparently unassuming, think of their work as supplementary to the physicians—superior to the opticians, and of definite service to the people. They claim no right of priority, nor that they possess the knowledge which is necessary to make an analytic study of the eye. They might be overoptimistic in believing they can safely, that is, quickly shift the responsibility of the care of the eye to the oculist. Regrettable as this is, unquestionably such men, however numerous, will continue their work for reasons often recited. We can find no fault here, and we could remain passive, and even cooperate with them when necessary. But the other group, class conscious, losing sight of the ease with which any of them, who have the necessary preliminary requirements, could acquire the knowledge to practice all they now make bold to demand, cooperate with, and lead a vast army of unprepared men and women along a dangerous path not in the interest of humanity. They would acquire knowledge of the human frame radiographically, as it were, thru the eye, admittedly a most wonderful organ, evidently one of God's happiest speculative ventures while considering the evolution of animal creation. Knowledge of it and its influence upon human economy comes only thru a knowledge first of the human frame, the fundamental study of which we thru years of experience worked out as a curriculum in our approach to medical practice. Let no man flatter himself that he is likely to serve well those who rely upon him, if he has avoided the path we have found so essential.

The use of the title doctor is purloined, and has lost much of its significance thru legislative enactment. The meek and lowly have become bold, and the public is at a loss to know "Who is Who." I am sure we all appreciate and respect the services of the opticians, who are a valuable adjunct to the practice of our speciality. The statutory doctor, however, who has grown aggressive and prosperous, now assumes the role of general custodian of the eyesight of the people.

When we read their advice to the public, we recall what Meredith said to Margot Asquith about Alfred Austin, "It is difficult to say what a bantam rooster has in mind when he is crowing." My own feeling toward these men is not personal, but is simply based upon the one thought, that it should not be made more difficult for the public to find a trained oculist.

The public finds thru experience, that the best educational opportunities which have been utilized is not in itself always enough, but it is the best protection the people have to safeguard them from exploitation.

We have a great society in the American Medical Association, but this organization can best function when the many problems concerning the public have been brought understandingly to its delegated authorities for solution and publicity.

There should be publicity from the right source of the right kind; then there will be less need to correct ill advised statements from any one. There is much sympathy with the suggestion made in the House of Delegates, at a recent meeting in Dallas, that Congress be requested to determine who shall use the title doctor. Medical education is on such a high plane at this time, that the young men who come out equipped for life service should feel that their honorable profession is reasonably protected from those who abuse the title given them.

We, who assume the education of our young medical men, must impress that numerous group in every graduating class who will have no interest in our special field with all the possibilities and abuses of the subject matter related. This can be a fruitful contribution in the interest of the people as well as of the great profession we would not only preserve, but make stronger and more useful thru the lives of those who have climbed the steep, narrow, beaten track, where Knowledge can be found with her handmaiden Humility.

DISCUSSION.

DR. JOHN E. FULTON, St. Paul, Minn.: The advent of the conscientious social service worker, in connection with the subject which we have under discussion, has aided us tremendously in acquiring knowledge of the cases as well as in applying successful remedies to ocular diseases. The importance of both are so well established in the mind of the profession, as well as the public, that the necessity of recommending them is superfluous. The oculist and the otolaryngologist simply constitute members of a group that can efficiently conduct this most important work. The oculist has the very great advantage over others, of being able to recognize early signs of pathology indicative of both local and constitutional disease in the living subject, and by the skillful use of the ophthalmoscope and other instruments of precision, detect changes in the fundus of the vessels and the optic nerve ending in their very earliest stages, thus giving most valuable assistance to his fellow associates in the way of correctly diagnosing diseases of the cardiovascular system, the kidneys, the brain and other organs.

A group of physicians, who organize themselves for this purpose, should be made up of highly efficient representatives of all of the specialties, and should have their findings referred to one head in order to avoid confusion in the mind of the patient. We often meet with patients, who have returned from some of our large clinics more confused as to their physical condition than before they applied for examination, on account of this lack of system.

A health standard will always be an imaginary point in the field that we are investigating. A correct health standard determined by more than one man will always be a variable quantity.

The doctor refers to focal points of infection, the subject which is more important in this line of work. I agree with George Woodruff that the conception of focal infection, together with the practical lines of treatment which it led up to, has been the greatest benefit to the human family that has been worked out in the last two or three decades. It was members of our own specialty who quickly recognized its vast importance and applied relief measures by virtue of knowledge thus obtained.

There is one point which we cannot emphasize too strongly, and that is that the minds of many specialists are so fixed on the tonsils and teeth as the place of focal infection, that the sinuses, respiratory tract, alimentary canal, prostate gland and other organs and glandular structures are only too frequently overlooked.

Great results have been obtained by our specialty in the establishment of periodic examinations in public school work. Before this work was commenced, vast numbers of children were neglected, so far as proper attention to the eyes were concerned.

The battle cry of practical therapeutics today is the early diagnosis of all diseases, and it is by means of these examinations that the commencing diseases of the eyes, and more particularly of the ear, are found in school children. In this work, the Child's Guidance Clinics, now being rapidly established in various parts of the country, aid us greatly in obtaining results heretofore not obtainable.

As to the general practitioner: I think most of them recognize the importance of periodic examinations being best made by a group of well selected specialists and the findings referred back to them for practical application. Team work in all difficult medical problems must necessarily be superior to individual examinations.

In regard to the relation of the dispensing optician and the trained oculist, it has always seemed to me to be a business proposition, the simple solution being that each one conduct his own financial part separately. I agree with Jackson that a truly expert optician has reason to be proud of his vocation, and it is essential to the success of the practicing oculist. The public, as well as the opticians, appreciate the importance of having all eye diseases referred to experienced and trained oculists.

The closing sentence of the essay in regard to the high plan of medical education, is well worth our most careful consideration, but it seems to be quite evident that the young physician, entering his field of work, will find himself quite comfortably protected by public opinion and really has but little to fear from the cults, as he will soon find, when he obtains a reputation in his profession, that members of the cults themselves will be among his patients when very seriously ill.

Dr. Horace Newhart, Minneapolis: Dr. Cary, in his very timely paper, has called attention to many points of great importance to the future practice of our chosen specialties. He has asked several questions of deep significance which cannot be answered by a simple yes or no.

I can best continue the discussion by asking still one more question, tho probably it is to sound a somewhat pessimistic note at this time. The question I would add to those already propounded by the essayist is, why is it that of late years the otolaryngologist and the ophthalmologist has been looked upon less as a wise and skilled practitioner of the healing art, and more as a mere artisan or technician, his general training and his medical interest in a given case too often being ignored or forgotten? This conception is becoming increasingly prevalent and is shared by both the profession and the laity. Among the latter there are today very many who are unable to distinguish between an ophthalmologist and an optometrist.

The reason for this condition we personally believe is due to overspecialization. We have been too prone to regard the special sense organs which constitute our field as isolated units of the body instead of as integral parts of the whole organism, and affected pathologically by disorders in even remote parts of the body. Thus we have in a measure divorced ourselves from the subject of general medicine, permitting others both within and outside the profession to assume on our part a lack of knowledge, or at least an indifference toward general pathology.

The periodic medical examination of the apparently healthy is rapidly becoming an important feature of American life. It is fostered and endorsed by every welfare organization, from the A. M. A. and the great life insurance companies to the local Rotary Club and Parent-Teacher Association. Its value is apparent to all. For the ophthalmologist and otolaryngologist, its growing popularity presents an unprecedented opportunity to prove where we stand in regard to the conservation of public health. We shall all of us be invited to contribute our part in the work.

We should not be slow to answer the call, but rather, by way of demonstrating our interest in the subject of saving the hearing and eyesight of our people, and with a reasonable reserve, should assume leadership in this work instead of a complacent detached aloofness, which has too long been our attitude in many communities. By wholehearted participation in the movement as individuals, and thru our endorsement and cooperation as societies, we may regain rapidly some of our lost prestige, and as broadly trained medical men demonstrate our genuine interest in the common welfare.

An especially large field lies open to us in otology. Recent examinations of the acuity of hearing among public school children reveals the existence of large numbers of neglected and even unsuspected cases of hearing impairment. Many of these cases, if discovered sufficiently early, may be saved serious hearing loss, if the active causes of their ear involvement can be discovered and removed. The solution of the problem of detecting slight hearing loss in large groups has lately been made possible. Recognition of the underlying cause and its removal often challenges the skill of the most expert diagnostician. In the proper evaluation of the possible contributing causes of deafness, the otolaryngologist has a great responsibility, insofar as these causes relate to the arrest of a further progress of the hearing loss, or the extension of ear disease. In meeting this responsibility, we have need for much specific and general medical knowledge, which in the past we may have forgotten or repudiated. The remedy for this situation must be found, in many instances, in retracing our steps and getting once more in close touch with every advance in medical knowledge which may have even a remote bearing upon our own narrower field. In other words, we must cultivate a broadness in our medical knowledge and point of view which will make us more efficient and will reinstate us in the minds of our conferes and the laity as well trained physicians whose opinion is worth while, and not as mere experts or artisans in a very limited field.

Dr. Jas. M. Patton, Omaha: I think we should emphasize one point in Dr. Cary's most excellent paper. We can say without egotism that we have, as a special group, information which is essential to the success of periodic health examinations. We have in the membership of this organization the most eminent and skillful teachers in the country, and to their undergraduates and to the men who come for postgraduate study, they are able to impress the necessity for careful preparation in this line of special work. They particularly impress upon their undergraduate students, that they must not attempt any of these manipulations or the care of ocular conditions without due preparation. But there are a great many men who have graduated and are in general practice, who will be called upon to make these periodic examinations, who may not have access to a specialist, and who will continue to carry on this work, so I feel that the rest of us must accept the responsibility of providing them with the necessary instructions. The essayist, our President, in his address yesterday, and the Chairman of the American Medical Association's Committee on Optometry, all have emphasized the fact that the optometrists are more than willing to assume this responsibility for the examination of the public's eyesight. Some of them, as has been pointed out, are very honest and capable, others perhaps not so

much so, but we can not complain and have no recourse whatever if the public, who are so enthusiastic over these periodic examinations, take the individual who apparently can give the best results. So I feel, gentlemen, that the responsibility comes back to us to teach the man in general practice, first, the fact that we think this is essential information; second, it is up to us to teach him, if he insists on doing certain phases of this special work, to do this work as satisfactorily and as expertly as possible. The objection has been raised, that if we teach the man in general practice who is doing a little refraction, perhaps doing it poorly, to refract correctly, we take money and patients away from our own individual practice and bank account. This is a most unworthy argument and fallacious as well. We are here to serve the public, to serve the general practitioner, and by so doing, as is always the case, we also serve ourselves. We can not hope to advance unless we are willing to contribute to this progress.

Dr. Hal Foster, Kansas City, Mo.: The first thing we must do is to convince the practical American people of the necessity of these periodic examinations. They have their machines and their watches examined, but it is hard to impress upon them the necessity of having themselves occasionally examined. During the late war, we were all very much surprised to find so many young men who had to be excluded from the service on account of deafness. I had charge of a Board in my town, and examined many hundreds of young men who were being recruited for the Army. There are a great many individuals now removing tonsils and doing sinus work, but they do only a part of their duty if they do not follow these cases up, especially children who have had some defects of hearing before the operation. Their work is only partly done if they do not look at the Eustachian tube occasionally; and if there is any defect of hearing, it should be attended to and the tube kept open. By doing that, they can correct these conditions and do these patients a great deal of good. It is almost too late in middle life to do much for patients. Our school boards realize that all over the United States, and they have the children examined as they enter school; then, if they have infected tonsils or sinus disease or adenoids, they should have this attended to before they enter middle life. In that way they prevent a number of these cases. We all know how easy it is to prevent deafness, and how very difficult it is to cure these cases after they get something the matter with them, especially children who have had scarlet fever, measles, mumps, or diseases of that character.

I want to emphasize all that Dr. Cary says, and I agree with him that we should keep step with the general internist and other physicians who are doing periodic examinations. We find, however, that we must convince people that they will get something out of it before we can accomplish much. The large insurance companies are doing that—urging periodic examinations, and also the great trunk railroads are urging that eyes and ears of their men be occasionally examined. We physicians should therefore keep this in mind and tell the people that it is absolutely necessary, and I certainly would emphasize the importance of examining children after adenoids are removed to see that deafness does not progress.

DR. HENRY MUNDT, Chicago: I am very glad indeed to have heard Dr. Cary's paper on this subject, because I think it is of immense importance. It is of extreme importance that the men doing specialties in this country consider this thing very seriously. There is, as Dr. Cary has said, a difficulty from the standpoint of the mental attitude of the general practitioner in making these examinations, and yet he is the man who will eventually make the examination. One difficulty with the general practitioner is that he is trained only to see sick people, and so after a period of years he probably has learned not to be interested in well people. If we are going to do what we should do in organized medicine for the general practitioner and for ourselves, we must take this thing up actively. One large commercialized institution in this country has conducted these examinations for several years and done it very well. We must absolutely get Medicine interested in this thing, or we will have it taken out of the hands of the practitioner of medicine. That is a very essential thing. How are we going to do it? If these examinations are going to be made, I would advise every man to follow the blanks put out by the American Medical Association. If you do that you will be much more apt to cover the ground properly than in any other way.

I do not think it is possible to make the very extensive examinations that have been suggested here—it is not even feasible from the standpoint of the patient. If the individual has gone to a fairly well trained practitioner of medicine, the outstanding thing will probably be found and he will be referred to the proper specialist to have the details worked out. Regarding laboratory examinations, I have for a long time thought that I could make a white and differential blood count, and a red blood count, a urine examination, and tell considerable about a patient, and I feel that any man who has paid any attention to that can do fairly well from the standpoint of infection, but there are many other features than infection which must be taken care of in these periodic health examinations.

We must also remember where the men who are doing the specialties sprang from. We came from the great body of medical men, and I make a plea to the men who are doing the specialties to support the medical men, support the local, county and state medical societies in their efforts to impress upon the practitioners of medicine every place the importance of periodic health examinations. The people are going to demand this. The Parent-Teachers' Associations, the Rotary Clubs, the organizations of that kind are interested in this thing. In our state the thing will be pushed and pushed very thoroly.

I am much interested in the talk about the optometrist, the optician and the oculist. Last year we tried a little experiment in Chicago. We had some cards printed with the three definitions on them of an oculist, an optometrist and an optician. I will confess that I had some difficulty in finding the proper definitions in the dictionary, but I finally found them. There are a great many people interested in that, and I think I never had anything in my office that was more satisfactory from the standpoint of answering questions about the relation of medicine to the oculist than these cards with the three definitions.

DR. A. A. HAYDEN, Chicago: I want to say that I am responsible for

the invitation extended from the Academy to Dr. Cary to read this paper, and I am largely responsible for his having prepared it, because he would not have done it unless I insisted that it be done. The reason for this was the fact that thruout the entire medical organizations of the country, periodic health examinations have been pretty fairly sold, but to my mind the specialist has been almost completely left out of the plan. It is our own fault. Dr. Newhart said we all should like to be called in to conduct these health examinations. His statement raised the question in my mind as to who was to issue this call. The general practitioners in our part of the country do everything from ingrown toenails to brain abscess, and only on rare occasions they will call a specialist.

One important question is whether the physicians will actually examine, as far as he can, the patients in his own particular practice, and whether he will refer these to the specialist at an early date—just when is the question that the individual examiner must decide. If he is honest, the patient will get a square deal. But which opinion is best about whether the tonsils are diseased or whether the sinuses need attention, whether the glasses fit properly, cannot in the majority of instances be accurately determined in the present state of our knowledge by the man doing entirely general practice.

I realize that there is some truth, and only some truth, in what Dr. Newhart said about the high priced experts. I believe that the high priced men are very frequently the cheap men. People are willing to pay twice as much money for a man to take out an appendix if he takes an hour to do it. They do not feel that they are so much overcharged as if he took only ten or fifteen minutes. It is a question, then, of educating the public to the fact of efficient medical service and where that service can be obtained, and most of all, that that sort of medical service is the cheapest in the end.

The point that I want to be obtained from this paper is the fact that we have been waiting to be asked into these examinations rather than having made for ourselves a place in the plan at the very beginning. I have often thought that the individual who is buying health examination should have recorded the findings of the physician, or the several physicians that make up that examination—should have recorded in a book of his own the findings that are made. They are easily forgotten if you do not do it. A plan of that sort, universally adopted, would lead to the accumulation of most vital statistics thruout the country.

But the purpose of the paper was to show that we have been asleep on the job, and if there is anything that can be done to arouse interest in the part that we can so ably play in this scheme, we should get busy and do it, because almost nothing has been done up to date.

Dr. R. H. T. Mann, Texarkana, Ark.: In order to fully appreciate Dr. Cary's very fine paper, the members of the Society must fully know its author. Dr. Cary, while practicing our specialty, has done far more than this. He is a great medical educator and a great business man in the great empire state of Texas.

Dr. Arthur J. Bedell, Albany, N. Y.: It seems to me that this problem resolves itself into one of education; first, of ourselves, which is always a most difficult thing to propose; second, the education of medical men generally; and finally, the education of the laity. When we reach the last group, our task will be very simple.

I feel that in the beginning of a campaign of this type, it is absolutely unwise to speak lightly of some one else, and in closing his discussion, I trust Dr. Cary will emphasize the point that we need education, but not at the expense of condemning the activities of others.

Dr. George W. Jean, Santa Barbara, Cal.: Regarding cults, my state is full of cultists, and we are fighting them by public health lectures given by the various specialists under the auspices of the Santa Barbara Cottage Hospital. The hospital gets the audience and does the publicity work.

Following Joslin's advice to specialists, I am having urinalyses done and fields taken in nearly every case. Why do ophthalmologists not have the fields taken, urinalyses made, eye pressure and blood pressure readings done in every case? We would find out a great deal and our patients would think us real doctors. A woman once asked me if I was a real doctor or just an eye doctor.

Dr. Cary was once a good judge of a poker hand, but his judgment of the size of a grape vine is poor. The largest grape vine in the world is at Carpinteria, California.

Dr. E. H. Cary, Dallas, Tex. (closing): I am very grateful for your kind words in regard to my paper. I have approached the matter from the angle of the specialist, and have tried to present this phase of the question in such a way that the average medical man would use the special knowledge he possesses.

I quite agree with Dr. Bedell, and I think the contents of the paper suggest that we need to educate ourselves on the subject. We also need to educate the majority of general practitioners. When we are alert and the general practitioner is informed, the next step is easy. The people themselves become educated when we are interested. My observation has been, that in things that are really worth while, we must lead the people. The masses still look to us for leadership and information. We have not lost out with the people. The trouble is that some men forget the principles which should govern us as practitioners, and some take pride in saying to the people whom they ought to treat, "I do not know anything about that—I do so-and-so." He may have been a well trained man, and as a matter of fact he is a well trained man; he knows a great deal about medicine, but somehow or other it becomes popular for a man to accentuate or emphasize his specialty and his particular information on the thing he is doing, and to discredit himself at the same time by saying, "I am doing so-and-so; I do not know anything about this." I think that is one reason why the people themselves do not believe that the men who are specialists have the broad training and the information which we know they do have.

I was not aware that there was any more beautiful demonstration of Nature's bounty than the great grape vine that we find near London, but you know, we have everything in this country better than in any other country, and when we stop to look around we realize that we have all things, cultivated or not, superior in California to products of other parts of the country. I am not surprised to be made aware at this moment by Dr. Jean that, altho the grape vine I mentioned is very beautiful, the one out at Carpinteria demonstrates the fact that God has done something in Jean's home State that man has not seen necessary to improve.

TRANSACTIONS

OF THE

THIRTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OPHTHALMOLOGIC DIVISION

A STUDY OF THE NUTRITION OF THE CRYSTALLINE LENS.*

The Cultivation of Lens Epithelium.

Daniel B. Kirby, M.D.

NEW YORK CITY.

It was proposed by Dr. John M. Wheeler, that an original investigation be conducted into the nature of the cause of senile cataract and the possibility of its prevention and arrest by nonsurgical means.

The literature contains a number of hypotheses as to the genesis of senile cataract, and records the various means that have been used in ancient and modern times to offset the formation of, or dissolve the opacity of the crystalline lens.

If such a complex biologic problem were approached from purely a clinical standpoint, and the study were based on observations and treatment of patients afflicted with incipient or mature senile cataract it was realized that the same difficulties that have conquered other investigators would be confronted and remain unsolved from a scientific standpoint. Nothing miraculous was expected, and it seemed that unless new standards were devised for the investigation of the cause and for judgment of the results of treatment, we would conclude with Jackson,[1] that the natural history of untreated cataract is so variable, that the results with simple hygienic care are as good as those that have been obtained with certain measures aimed directly at the solution of the opacity of the lens.

It is not too much to expect that some means of influencing the nutrition of the lens in vivo may be found. Since the ad-

* The basis of this report is derived from work conducted by the author as Research Fellow for the American Academy of Ophthalmology and Oto-Laryngology, and by virtue of a grant secured from an anonymous donor by Dr. John M. Wheeler. The experiments were carried on by the Department of Ophthalmology in the Departments of Anatomy, Physiology, and Chemistry at the University and Bellevue Hospital Medical College. Acknowledgements are gratefully made.

vent of the use of the slit lamp and microscope, many opacities and areas or zones of increased relucency of the lens have been found in apparently normal eyes with normal vision. Covered by transparent cortex, they are mute evidence that the lens in point suffered a physiologic insult at one time, and was able to survive and recover, and to bury the opacity by the formation of clear cortex. Cataract may be a deficiency disease, and it may be possible to discover the nature of the deficiency.

It was decided to approach the problem from a scientific rather than an empiric standpoint, and to go back to fundamentals. A study of the developing lens in the living embryo, and of the living cells of the lens offered possibilities. It was also thought, if the cells of the lens could be cultivated in vitro, that here would be a new tool, a new mode of expression, a new standard for our investigation into the nutrition of the lens. If the study of the cause of cataract could be narrowed down to the life and death of the single cell derived from the lens, a great step forward would have been taken. The single cell is not so simple, but it is far simpler than the complex lens organ. It would form a basis for new experimentation, and by means of this, it might be possible to analyze old theories and test out new ones.

Tissue Culture.

Since the discovery by Harrison,[2] in 1907, that cells could be caused to migrate and divide outside the body, various investigators have used this method for the study of morphologic and physiologic problems.

Carrel[3] developed an ingenious technic, by which permanent pure stains of fibroblasts could be isolated and cultivated in vitro. He established the procedures by which the method of tissue culture is becoming adapted to its main object, the study of the fundamental problems of physiology and pathology. The early technic, consisting of placing a fragment of tissue in a hanging drop of culture medium, did not allow an accurate analysis of the action of a tissue upon other tissues and upon the humors. The cells were subjected to complex influences, such as those of necrotic cells of their own type, of living and dead cells of their types, and of a medium which deteriorated spontaneously in a short time.[4]

Advances have been made during the last few years, chiefly thru the embryonic extract plasma clot method of Carrel, by which those difficulties and objections are overcome, and have contributed markedly to the progress of an important method of investigation, primarily by the isolation of pure strains of cells,[5] and by the development of procedures for measuring the rate of growth and for studying the functional activity of the tissues.[6,7]

Fischer[8] isolated an unmixed strain of epithelium from the anlage of the chick iris in 1922. Ebeling[9] produced cultures of the thyroid epithelium in 1925. It has been possible to carry these cultures on for long periods of time. It has been proved that the cells remain true to type, that they do not dedifferentiate, and that they produce their typical substances in vitro.

In the experiments about to be reported the method and technic of Carrel* ([3, 4, 5, 6, 7]) were used.

Experiment Demonstrating the Metamorphosis of the Lens in Vitro.

The eyes of two chick embryos of about 52 hours incubation (lens vesicle stage) were removed, and the anterior one-third of each eye placed in a homogeneous medium consisting of equal parts of adult chicken plasma and chick embryonic extract. The concave slide method of Carrel was used. The explant was oriented so that the lens vesicle in each could be plainly observed with the microscope. They were allowed to incubate for 72 hours at 37.5 degrees centigrade, and in two of the specimens it was observed that differentiation of the posterior portion of the lens vesicle had occurred, the cells having elongated to form lens cortex. In the other two, due to digestion of the medium, the specimens were not in proper position for observation. Microscopic sections of the specimens were not obtained. Since this work, the author has learned of the explants of the entire eyes of amphibian embryos by Filatow[10] and of chick embryo eyes by Strangeways and Fell.[11] The latter workers demonstrated by microscopic sections the differentiation of the eyes, which progressed in vitro in a surprisingly normal way.

*Acknowledgements are gratefully made to Drs. Alexis Carrel and A. H. Ebeling for their many courtesies.

The Growth and Cultivation of the Crystalline Lens Epithelium in Vitro.

Fischer[8] made numerous explants of the lens with the epithelium of the anlage of the chick iris. In his report, he recorded his observation that the lens tissue did not grow at all.

It appears necessary therefore in reporting the cultivation of lens epithelium in vitro to be able to definitely prove the identity of the tissue explanted. This will be done: (I) by repeating Fischer's[8] experiments on the iris epithelium to show that if extraneous tissue is explanted with the lens, the extraneous tissue will grow, and if the explant of the lens also grows, one will contaminate the other; (II) by proving that proper technic of microscpic dissection of the embryo lens frees the lens from all extraneous tissue; (III) that the epithelium of the lens so freed and properly cultivated in vitro will migrate and proliferate; and (IV) that secondary and tertiary cultures from these can be made, so that a strain of pure lens epithelium can be developed.

Experiment No. I. The lens and vitreous of chick embryos of from four to nine days incubation at 37.5 degrees centigrade, were removed by incising the posterior portion of the optic vesicle and using traction on the vitreous. Usually a ring of pigmented and nonpigmented epithelium of the rim of the optic vesicle comes away with the lens and vitreous. If the vitreous be excised and the remaining explant cultivated in the manner used by Fischer,[8] a growth of epithelium from the iris anlage is obtained.

This experiment demonstrates that the epithelium of the iris anlage can be cultivated in vitro, and, also, that if an unmixed strain of lens epithelium is desired, all extraneous tissue must be removed from the lens. Strains of iris epithelium were carried along in successive subcultures, so that a ready comparison with the cultures of the lens epithelium was always available.

Experiment No. II. After many experiments, it was found that the eyes of chick embryos incubated for five days at 37.5 degrees centigrade were ideal for dissecting the crystalline lens free from all extraneous tissue. The eyes usually measured 3 mm. in diameter, and the lens slightly less than 1 mm. in equatorial diameter. Both prior to this stage and after it, the protoplasmic adhesions are such as to make it almost impos-

sible to express the lens free from other cells. The embryo is placed in a concave slide, and the amnion removed from the eye region. A sharp, cutting needle in a holder is used in each hand, one to steady the eye and the other to incise the posterior portion of the eye and draw out the vitreous on the point of the needle, bringing with it the lens and a portion of the rim of the optic cup. This specimen is placed in Tyrode solution[12] in a clean concave slide, under the binocular dissecting microscope, using oculars 2 and objectives A2 with transmitted light. The needles are replaced by fresh ones. These are inserted between the equator of the lens and the investing tissues, and the other needle gently expresses the lens free from all extraneous tissue. This is transferred to fresh Tyrode solution and then to the embryonic extract plasma clot medium. After 72 hours incubation, it was observed that no outgrowth of cells occurred.

This experiment demonstrates that it is possible to dissect out the crystalline lens of a chick embryo of five days incubation in such a manner as to free it from all extraneous tissue, and that the lens epithelium is contained within a cuticula outside of which, when uninjured, the cells do not migrate. (Salzmann[13] found that the lens vesicle is soon closed off from the neighboring structures by a cuticula, later the lens capsule.)

Experiment No. III. Lenses from five day chick embryos, isolated in the manner described in Experiment No. II, were incubated in toto for 24 hours at 37.5 degrees centigrade. The cultures were then opened, and the lenses removed from the media and divided into two pieces. Outgrowths of cells were obtained from the epithelium of two of four lenses incubated. One of these, No. 667-2, represented the beginning of a strain of lens epithelium which will be described in Experiment No. VI as Strain C.

Those incubated for a longer period apparently retained no viable cells, as no outgrowths of cells were obtained after section. Some of these specimens took up and retained the neutral red vital stain (1-30,000) during the first period of incubation. The trauma of the expression of these particular lenses may have had something to do with their inability to withstand cultivation outside of the body. Undoubtedly the cuticula of this stage (later the lens capsule) has much to do as a semipermeable membrane with the process of nutrition

of the embryonic lens, and a slight injury to this membrane would destroy the possibility of the lens living in vitro.

Twenty-four hours is sufficient time for incubation of tissues from a five-day embryo to exclude any growth of cells extraneous to the cuticula of the lens. In our experiments on tissue from this age embryo, if growth is to occur, it will be evident after twenty-four hours.

This experiment demonstrates that it is possible to rule out tissue extraneous to the cuticula of the embryonic lens by cultivation, and then by section of the lens to obtain a culture of an unmixed primary culture of epithelium from the interior of the embryonic lens.

Experiment No. IV. Cultivation of Lens Epithelium in vitro. Lenses of five day embryos were expressed on numerous occasions in the manner described in Experiment No. II. Having been placed in fresh Tyrode solution, and again replacing the needles for fresh ones, the lenses were incised anteriorly and the lens epithelium stripped off easily to the equatorial region, as there exists a potential space at this stage between the lens epithelium and the cortex. The lens epithelium is then explanted on the point of a needle directly to the embryonic tissue juice plasma clot medium. The cortex is explanted into another slide. Incubated at 37.5 degrees centigrade.

After twelve to twenty-four hours, the lens epithelium begins to migrate, divide by mitosis and multiply. After forty-eight hours, observations are recorded. Apparently the entire explant lives, but cells do not wander out from all parts. This depends on the particular conditions of the explant, foldings, the exposure of the cells, the trauma, and the conditions of the media. Many bizarre shapes have been observed. Small pieces of epithelium do as well as large ones. Cells, apparently temporarily united, move out from the explant, never losing contact, however. The walls of cells in apposition are difficult of definition in the living cultures, but apparently the cells do not form a syncytium. Both membrane and peninsula formation are observed. The explant remains transparent, and the newly formed cells are transparent. The wall of the healthy cell is a geometric line. Protoplasmic processes of various lengths are seen, some being very long. The cytoplasm is clear, containing granules, some of which take up neutral red in weak solution, while others do not. The mito-

chondria are visible in the unstained cells. When the microscope substage diaphragm is opened and slight excess light admitted, the cells become invisible. The nucleus is ovoid and usually contains one or two nucleoli. All stages of mitoses may be observed. About the entire living culture, there is observed a halo of transparency of the media, indicating either the influence of the cell products on the media, or the preparation of the media by the cells for their nutriment. The outgrowth of the cells is apparently preceded by a process of digestion of the media.

Fresh explants of lens epithelium should be cultivated on No. 0 glass coverslips and examined with the aid of high magnification, to prove the absence of any contaminating cells. Fibroblasts can be differentiated, and the living pigment epithelial cells of the eye, if present, will show definite, completely opaque, rod shaped, ovoid or rounded pigment granules. The nature of these cells from the iris anlage, and their appearance when cultivated in vitro, must be thoroly studied by anyone attempting to cultivate lens epithelium.

The explants of cortex do not grow. No cells wander or divide. Partial solution of the explant takes place by a process of hydrolysis. Disintegration of the cells occurs, and the explant has a fibrillated, washed out appearance. This process can best be observed by allowing the cortex to remain in Tyrode solution, and observing it with the microscope. Hydration and swelling occur quickly; a beaded appearance is produced by globules forming in the cells. These globules rupture, the cell bursts and the cytoplasm goes out into the solution, which is isotonic for other tissues but hypotonic for lens cortex. A solution containing 1.2% NaCl is isotonic for the lens cortex, in that this hydrolysis does not take place, but cell migration and division do not occur. Apparently, these cells are no longer capable of division—are biologically old even at this early stage of differentiation. They have a different osmotic coefficient than the anterior lens epithelial cells.

This experiment demonstrates, that lens epithelium, when cultivated in vitro under proper conditions, will live, migrate, divide and multiply, and that under these same conditions, the lens cortex cells will not do so. A distinction is drawn between lens epithelium, which is limited to the anterior and equatorial regions of the lens, and the lens cortex, which constitutes the differentiated bulk of the lens. They are both epi-

thelial. From the anatomic arrangement of the cells, the anterior epithelium and their elongation at the equatorial region passing over into the lens cortex, it may be safe to assume that the cells of the cortex are derived from the epithelial layer, or at least from those cells which are at the equator. It may be possible to grow the cells of the cortex, as long as they retain their nuclei, under different conditions of cultivation. Alterations have been made in the media, but no successful cultures have been obtained. These facts, if substantiated by further experiments, would lend proof to the idea that the anterior epithelium of the lens differentiates into the cortex.

EXPERIMENT No. V. Extract of nine day chick embryo eyes was used in place of extract of the whole body. It was made by grinding the eyes in a Wedgwood mortar with pestle and alkali free ground glass. It was diluted with Tyrode solution and centrifuged. The explants grew well when this was used in place of the whole body extract, but were not free from retinal pigment rods in the cells. Now, either pigment cells had been explanted with the lens, or the cells were transferred with the eye extract, or the pigment rods in the eye extract were ingested by the cells. It has already been demonstrated that the lens can be explanted free from pigment cells. The media without explant was cultivated and proved sterile and containing no cells. It was difficult to centrifuge down all the pigment rods, and these could be seen with the aid of oil immersion lens. It was, therefore, determined to use extract of the embryo body minus the eyes. This has been done, and transparent growths free from any retinal pigment rods were obtained. Dr. A. H. Ebeling has been kind enough to examine some of our cultures, and has found no contaminating cells in one produced by our perfected technic.

This experiment demonstrates that lens epithelium can be cultivated in media containing extract from the embryonic eye alone, and also from the embryonic body minus the eyes. In the latter media, the cells can be proved free from any contaminating cells.

EXPERIMENT No. VI. To produce an unmixed strain of lens epithelium which can be carried along indefinitely and used for controlled experiments.

Living explants from lenses isolated and cultivated as described in Experiment No. IV., can be divided and two subcultures obtained. These can in turn be subdivided after cul-

tivation, and thus secondary and tertiary cultures obtained. By this means, a strain of lens epithelium can be propagated from an original single explant. One of our strains (C) was obtained as described in Experiment No. III, after having incubated the entire lens, proving the absence of any cells extraneous to the cuticula. The history of this strain may be reported as exemplifying the culture of lens epithelium under these conditions.

June 28, 1926 (No. 667-3). Entire lens explanted from a five day embryo.

June 29, 1926 (No. 669-2). The lens remains transparent. Dissected out of the media. Cortex shelled out. Found granular. Partially distintegrated. Epithelium subcultivated.

July 1. Several processes. Cellular outgrowth from explant.

July 3. Small growth, vacuolated cells. Media changed.

July 6. Good growth of cells. Membrane and peninsula formation.

July 7, 9, 12, 14, 16, 19, 21, 23, 26, 28, 30, Aug. 2, 4, 6. Media changed and culture divided and subcultivated.

One-half of the culture was put on a mica coverslip, and one-half on a glass coverslip. Latter examined on the ensuing working day with oil immersion lens, and then fixed and stained for further study. The culture on mica was used for the propagation of the strain. Extract of the embryonic body minus eyes was used thruout. The divided cultures about double in size every forty-eight hours. The cells are transparent and remain epithelial in type. They do not dedifferentiate.

This experiment demonstrates that lens epithelium cultivated in vitro can be carried on for successive generations Strain C. was carried thru seventeen generations, over a period of six weeks, and was thriving when the work was discontinued, August 9th. It was then ready for controlled experiments and observations on the rate and character of the growth, the cellular reactions and the substances produced by the cells.

Discussion.

In this study of the cause of cataract and the possibility of its prevention or arrest by nonsurgical means, the problem

has been approached from the scientific side, namely an experimental study of the nutrition of the crystalline lens. The first step forward has been taken, that of the cultivation of an unmixed strain of lens epithelium in vitro.

It is hoped that these cells from the chick embryo form a valid means for search into the complex biologic problem of the nutrition of the lens in homo:

1. Because, even at the very early embryonic stage at which the cells are taken, differentiation has progressed in the lens, so that the cells of the cortex are already biologically old. The nuclei of these cells stain less densely, the cytoplasm has acquired a different biochemic composition than the ectodermal cells from which they are derived. The cells are progressing toward the adult nonnucleated lens fiber. They have the same osmotic coefficient[14] as the adult lens fiber.[15]

2. Because the metamorphosis of the eye is accomplished in a nonspecific manner. Uhlenhuth[16,17] has shown that the metamorphosis of the eye is dependent on substance or factor furnished by the embryonic body, and that this factor can be supplied not only by an embryo of the same species, but also by one of a different species.

3. Because the origin and early differentiation of the lens is accomplished in a nonspecific manner. Lewis,[18] by experiment, proved that the lens in its normal development is dependent on the contact of the optic vesicle with the ectoderm. This can be accomplished not only by the optic vesicle of the same species, but also by bringing about the contact of the optic vesicle of one species with the ectoderm of a foreign species.

4. Because the adult lens contains an organ specific protein.[19] It gives the same immune reactions with a sensitized serum whether the protein is from the lens of mammals, including homo, amphibia, birds or fish. There is some evidence that the embryo lens contains this organ specific protein even at an early stage.[20] More work must be done on this before it can be proven definitely.

The lens grows constantly thruout its normal life. The cells are not cast off, but are constantly being compressed and by a process of sclerosis forming the nucleus of the lens. This growth energy of the lens epithelium in vivo may be expressed as a function of the concentrations of the growth producing and the growth inhibiting substances taken up by

the lens. There must be a delicate balance between the two factors to maintain the normal anatomic relations. Tissues in vivo may continuously receive substances in very high dilutions.[21] It may or it may not be possible to detect the presence of such substances in vitro. Can it be, that there is such a substance which has to do in a specific manner with the growth and differentiation of the crystalline lens thruout its normal life? If so, is this substance produced within the eye or elsewhere in the body?

Transparency of the tissue of the lens is brought about by a certain physicochemical combination of protoplasm, that changes the index of refraction of the cells. The secret or principle of this is contained within the living transparent cells of the lens waiting to be discovered. These cells have a peculiar composition, such as is found nowhere else in the body. The osmotic coefficient is different; the water binding power of this protein is unique. Are the earliest signs of incipient cataract as observed with the aid of the slit lamp and microscope, namely, lamina separation, formation of globules and vacuoles, and water splitting of fissures but an indication of changes in the protein of the lens affecting its water binding power?

These are some questions to which an answer must be found before further progress can be made in the study of the nutrition of the lens and the cause of senile cataract.

Conclusions.

1. Differentiation of the posterior cells of the lens vesicle into lens cortex has been observed in vitro.

2. It is possible to dissect out the lens of the five day embryo chick in such a manner as to free it from all extraneous cells.

3. Lens epithelium even at a very early stage is contained within a cuticula.

4. Lens epithelium under proper conditions will live, migrate, divide, and multiply in vitro in primary explants.

5. An unmixed strain of lens epithelium can be propagrate, divide, and multiply in vitro in primary explants. This strain can be utilized for controlled experiments on the nutrition of these lens cells.

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DISCUSSION

DR. W. B. LANCASTER, Boston: I congratulate Dr. Kirby on the notable contribution he has made to ophthalmology. He has done a difficult piece of work and done it well. I wish to thank Dr. Wheeler, for it was thru him that the committee was able to secure Dr. Kirby as research worker. He suggested the subject to be studied, and, indeed, without the financial support which he made available, the work could not have been done, for the Academy stipend alone was quite inadequate. I might point out that this work of cell culture requires a vast expenditure of time and skill. Dr. Kirby has kept two technicians at work, and he was able to secure a highly skilled and experienced technician, who had done a great deal of work of this sort at the Rockefeller Institute. Besides the advice

and support of Dr. Wheeler, Dr. Kirby had the benefit of close relations with Dr. Carrel and Dr. Ebeling at the Rockefeller Institute, where the most noteworthy work on cell culture has been carried on and the technic developed. They have been keenly interested in Dr. Kirby's work and regard it as a highly commendable achievement.

Under the circumstances, it was a source of no little satisfaction and pleasure to the chairman of the committee on research, that Dr. Kirby was eager to have his investigations carried on under the auspices of the Academy in preference to other attractive alternatives.

I spoke of the time consuming character of the work and the skill required. The cell cultures grow so fast, that they have to be transferred to fresh media every two or, at most, three days. This must be done with perfect aseptic technic, for the media are excellent for microorganisms, and if they invade the cell culture, it is ruined. The minute size and the fragility of the growths to be divided and transplanted, the necessity of having the media perfect, not only in asepsis, but in chemical composition and freedom from all extraneous substances, the importance of maintaining proper temperatures, and finally, the necessity of keeping full and accurate records, and the preservation, staining, and mounting of slides—these are some of the things which occur to me, but I spent only a few hours in the laboratory, and my knowledge of the subject is very fragmentary and mostly derived from the few articles I have read. I am sure there are many other details that I have omitted which have to be looked after, and so make demands on time and skill.

In carrying on cell culture, it is necessary, first, to isolate the cells it is desired to study, and they must be in pure culture uncontaminated by other cells; second, to get and maintain a suitable medium of unvarying composition. When tissue cells were first made to grow in a hanging drop of some suitable medium, they did not long survive, because of various more or less obscure influences, such as living and dead cells of the same and of other types in the tissue, and such as deterioration of the medium by losing some and gaining other constituents, thus becoming unfit.

When these two conditions have been fulfilled, viz., isolating the cells and providing and maintaining a suitable medium, it is then possible to study the rate of growth of the cells and how it is influenced by (a) the presence of other cells, (b) the presence of chemical substances, (c) the absence of chemical substances.

The first cells to be isolated were fibroblasts, because they are the easiest. Almost any tissue will provide them, and they outgrow all other cells and so are soon in pure culture. Strains have been grown for over a thousand generations. Many studies have been made of factors influencing the growth of fibroblasts, some activating like embryonic tissue juice, and others inhibiting.

Many futile attempts to isolate epithelium were made. Always fibroblasts outgrew and crowded out the epithelium. Finally, Fischer in 1922 succeeded by utilizing the epithelium of the iris, which adheres in minute but sufficient quantity to the lens when the lens is removed from the eye of a chick embryo.

Epithelial cell cultures double in size in three days, while fibroblasts double in two days. At one time, it was thought that epithelial cells tend to dedifferentiate in time into connective tissue. But the real explanation of the change from epithelium to connective tissue is that the fibroblasts

crowd out and displace the epithelial cells in the cultures. When Carrel in January, 1924, published his review from which I am quoting, no strain of thyroid, renal, or other epithelial cells had been obtained. Corneal epithelium had been grown for a brief time. Cartilage cells had been grown, obtained from the embryo eye of the chick (pars cartilaginis sclerae). There had been success with large mononuclear leucocytes (macrophages). At first, polymorphonuclears and leucocytes grow, but these gradually disappear and soon only macrophages are left.

This was the stage of progress when Dr. Kirby started. The device employed by Dr. Kirby to make sure that his explant was free from extraneous cells, which are so likely to spoil the experiment, was very ingenious. I refer to his incubating the specimen twenty-four hours, to establish the fact that no viable epithelial or fibroblast cells from the iris or elsewhere were present on the outside of the lens before he opened the capsule to make an explant of the lens epithelium.

The importance and future value of cell culture is not appreciated at first glance, but after reading some of the literature and giving rein to your imagination, the possibilities grow on one. Carrel, who is very careful not to overstate the case, says that it is probable that the study of pure strains of cells and of their interactions with the humors of the organism will play as important a part in the progress of physiology as the use of pure cultures of microorganisms in the development of bacteriology.

For example, it has been shown already that epithelial cells do not build up protoplasm from serum. Serum, in fact, has a depressing effect on the proliferation of epithelial cells, and this effect increases markedly with the age of the animal. Lymphocytes and large mononuclear leucocytes can live in serum and manufacture from its constituents growth promoting substances. They are capable of being nurses of epithelial cells and of fibroblasts. This illustrates how cells of one sort act on other cells. Besides growth promoting substances, there are growth inhibiting substances. The necessity of supplying one sort and furnishing means to carry away the other sort, or to prevent its action, is apparent in the problem of growth and the health of the cells. The amazing spectacle we have just witnessed on the screen is enough to impress on us the supreme importance of control. We have seen the vigor with which the cells carry on their activities. The colonies of cells double in one to three days when conditions are favorable. But unless a transplantation is done at frequent intervals, the growth soon stops, due to accumulation of what should be carried off in the lymph and blood streams in the living animal, and the exhaustion of materials which would be supplied by the lymph and blood streams. The study of the factors which control or regulate growth is seen to have an importance which it is hard to exaggerate.

"The important thing is, that it has become possible to detect in the humors of the organism and other fluids the presence of certain substances which act directly on cell nutrition. The technics are still in the early stages of their development, and must be used with great care to yield accurate results." (Carrel.)

The road is a long and difficult one, but what a thrill those workers have whose privilege it is to lead the way around one more obstacle and clear the road for the rest of us. We speak of the *secrets* of Nature, but really Nature is not secretive. She stands ready to reveal all to the understanding mind.

Figure 1. Fresh explant of lens epithelium. Zenker Acetic. Harris Hematoxylin. x 220.

Figure 2. Strain of lens epithelium. x 60.

Figure 3. Cells from strain of lens epithelium. Zenker Acetic. Harris Hematoxylin. x 450.

Fig. 4.

Fig. 5.

Figure 4. Cells from strain of lens epithelium. Zenker Acetic. Harris Hematoxylin x 600.

Figure 5. do x 600 showing herniations of the cytoplasm. Degenerative change.

Figure 6. do x 450.

Fig. 7.

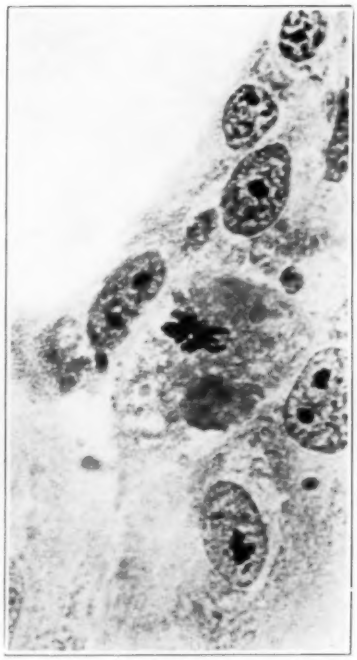

Fig. 8.

Figure 7. Mitotic figure in strain of lens epithelium. Zenker Acetic. Harris Hematoxylin. x 450.
Figure 8. do.

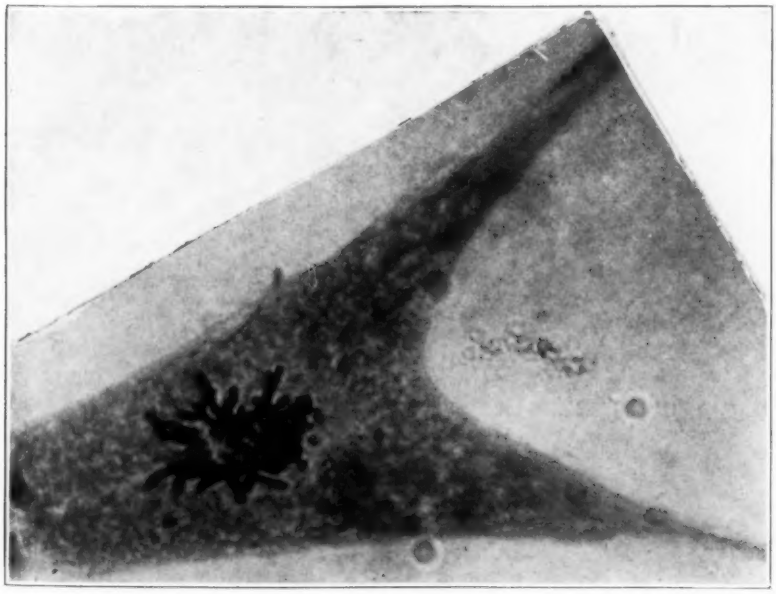

Figure 9. do x 1000.

DIAGNOSIS AND TREATMENT OF VERTICAL DEVIATIONS OF THE EYES.

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The object of this paper is to emphasize the following facts:

A. In the diagnosis:

1. A hidden vertical deviation is the commonest cause of a persistence of asthenopic symptoms after accurate correction of a refractive error and any apparent heterophoria.

2. A complete and accurate diagnosis of heterophoria, in presence, kind and degree, can be made only by the aid of prolonged monocular occlusion (Marlow). This procedure is the same to the extraocular muscles as cycloplegia to the intraocular, and its importance can be upheld by the same line of reasoning.

As absolute proof of these statements, Table I, of 110 cases that tested exact orthophoria in all directions, before occlusion, is submitted. In these cases, the final tests as regards vertical deviations only, were as follows (in prism degrees): 1° to 2°, 28 cases: 2° to 3°, 32 cases; 3° to 4°, 18 cases; 4° to 5°, 13 cases; 5° to 6°, 3 cases; 6° to 7°, 2 cases; 7° to 8°, 2 cases; 9° to 10°, 3 cases; 10° to 11°, 1 case; 12°, 1 case.

3. Nearly all cases of vertical deviation show a slight paresis of a superior rectus on making the diplopia tests, in rotations, after the full deviation is developed by occlusion.

B. In the treatment:

1. In the lower degrees of vertical deviations full correction, by prisms, is usually effective in giving relief.

2. In cases beyond the possibility of prism correction, or when, because of low refractive error, the patient wishes to avoid altogether the wearing of glasses, operation can be done, by my shortening method, applied to the weak muscle, without any fear of bad results.

Stevens was of the opinion, that one degree of vertical

deviation could cause symptoms as severe as fourteen of horizontal. There is no doubt that it does cause disproportionately severe symptoms. I have one case in which the correction of two degrees hyperphoria (hidden) caused the disappearance not only of headaches and disabling eye symptoms, but also of digestive troubles so severe, that an exploratory abdominal operation, to determine their cause, was under serious consideration. The reason for this disproportion is, in all likelihood, that the correction of a vertical deviation involves an entirely abnormal use of the muscular apparatus. Either the elevators of one eye act with the depressors of the other in varying amounts to bring the eyes to a level, or one group acts for the entire amount. In either event, the functioning is abnormal as compared with the more or less normal action of the horizontal recti in overcoming horizontal deviations. Furthermore, horizontal deviations, especially esophoria, are often spasmodic (false) and lessen or disappear on correcting the refractive strain, while a vertical deviation is usually true and permanent. The results of monocular occlusion, given in Table I, prove this statement.

Prolonged Monocular Occlusion.

I have been using this procedure since early in 1919 and, in a short paper read before the Pacific Coast Oto-Ophthalmological Society in that year, I emphasized its importance. By the spring of 1920, data in about 50 cases had been accumulated, with the idea of publication, when Marlow's paper appeared in the British Journal of Ophthalmology. Until then I had been ignorant of his work. In my opinion, he is entitled to credit for the discovery of a procedure second only to that of the correction of refractive errors. I never treat a symptom causing heterophoria till the information afforded by monocular occlusion is at hand. I have seen so many cases of esophoria disappear, to be replaced by hyperphoria, that I now suspect such cases to be spasmodic conditions of the interni associated with the effort of bringing the eyes to the same level.

My first case was diagnosed accidentally, being a young woman who had had headaches and eye disability for many years. She was practically emmetropic, and showed an esophoria of only four degrees. All treatments except operation had been

tried, and she was referred to me for that purpose. It had always been my habit to follow Worth's plan of occluding one eye before operation, in order to develop the total deviation. This was done in expectation of finding sufficient esophoria to warrant operation. Much to my surprise, the esophoria disappeared, to be replaced by exophoria of four degrees and a hyperphoria of the same amount. She was perfectly comfortable while wearing the patch—the first time in many years. She was given vertical prisms and her troubles were over.

Since that experience, I have employed occlusion in all cases where it appeared that the symptoms were due to the eyes, and where proper correction of any refractive error had failed to give relief. My total number is now approximately 1,500, while in the same time I have had about 5,000 new patients of all kinds. It is apparent that I would have missed the diagnosis in easily one-third of that class that consulted me for an ordinary examination leading to the giving of lenses.

A more general use of this procedure will reduce almost to zero the number of cases that are ordinarily called "retinal asthenopia."

In addition to the true muscle balance, the following information is obtained by the occlusion test:

1. Whether or not the patient is really suffering as much as claimed. I always tell my patients that this is one of my reasons for advising its use.

2. The character of the deviation, whether paretic or not, by making the diplopia tests in rotation. It is most important to locate the weak muscle, for, in case the deviation is enough to require operation, that muscle should be shortened. I would consider myself guilty of malpractice in tenotomizing a right superior rectus to balance a paretic left superior rectus.

3. Whether or not any imbalance found is the cause of the symptoms. In practically all such cases, the discomfort and disability disappears, or is noticeably lessened, when the binocular strain is abolished, and this in spite of the annoyances incidental to monocular vision. For this purpose, the patch is worth while in cases of pure insufficiency of convergence, of which I have seen many.

In his summary for the literature for the Year Book, Dr. Duane raised the objection that normal eyes might show these deviations if occluded. This is perfectly true as shown by my table. Some objectors use the same argument as optometrists

against cycloplegia in the diagnosis of refractive conditions. Many ametropes have no symptoms, and we all know that many people have high degrees of muscle imbalance and no trouble. The following facts prove that the measurements disclosed by occlusion are true deviations:

1. Prisms prescribed on the basis of the patch measurement do not cause the appearance of an opposite test.

2. Cases operated on the same basis do not develop the opposite condition, even when occluded later on to determine this point.

In using monocular occlusion, the sighting eye is located by the ring test, and the patient directed to keep the other occluded all the time he is awake. He is requested to use the one as he would like to use the two, and to note whether or not the discomfort lessens. The relief is often marvelously prompt, as shown by the case mentioned early in the paper. Measurements are to be taken every two days till two agree. I have had cases where, on first test, no appreciable change had occurred, but the relief afforded caused it to be continued, with success in developing a suspected deviation. Most of the cases take six days, some eight, and a few ten to become consistent.

Changes after patching occur in approximately the following order of frequency:

1. Increase in a present vertical deviation or the appearance of a hidden one.

2. Increase in divergence shown by:

(a) Increase in a present exophoria.

(b) Orthophoria may become an exophoria.

(c) Esophoria lessens.

(d) Esophoria disappears to be replaced by exophoria.

3. Appearance of a combination of exophoria with the vertical deviation.

4. Increase of an esophoria (uncommon).

5. Reversal of vertical tests (rare).

The commonest test before occlusion is one of low esophoria, only, which changes almost constantly to exophoria with hyperphoria. It is worth while to call attention to the frequency of insufficiency of convergence.

In placing a value on Table I, the following points must be considered:

1. All of these cases had severe symptoms, amounting to practically complete disability from eye fatigue, headaches or both. Many had carsickness, dizziness. Some had nausea and digestive difficulties. A common complaint was pains in the neck, and many were head tilters.

2. Most of them had had many examinations with no relief. Those having first examination failed to show enough, by the usual method, to account for the severe symptoms. Some were even emmetropic.

3. The results column proves the value of the method in the almost uniform relief.

4. Most of those who failed to obtain relief had too complicated a deviation to correct by prisms, or had a definite insufficiency of convergence. Some of these permitted operation and secured the desired improvement.

5. Good results cannot be secured unless one knows what he is treating.

6. Under the other ideas, tenotomies for nonexistent esophoria could have been done in many cases.

7. The conclusion is unavoidable, that the monocular patch is a necessary preliminary to the intelligent treatment of the heterophorias, and that good operative results, in the absence of the information it affords, are apt to be due more to good luck than to good management.

SUMMARY OF TABLE I.

Degrees of deviation	Vertical deviation	Exophoria	Esophoria	Lateral orthophoria
1— 2.....	28	13	1	14
2— 3.....	32	10	1	..
3— 4.....	18	13	..	..
4— 5.....	13	19	..	..
5— 6.....	3	11	..	..
6— 7.....	2	11	1	..
7— 8.....	2	3	..	..
8— 9.....	..	7	..	..
9—10.....	3	3	..	..
10—11.....	1	..	..	..
11—12.....	..	1	..	..
12—13.....	1	..	..	..
13—14.....	..	2	..	..
Totals.....	103	93	3	14—110

Treatment.

A. By prisms:

1. Bases up and down. A full correction of the vertical deviation is usually accepted. It is divided equally between the two eyes, except occasionally when the condition is pronounced paretic. In this event, it is made stronger in one eye, with the idea of approaching a normal balance on looking down—the reading position.

2. Bases on the oblique. In cases where the vertical deviation is small, and there is a horizontal one of some moment, the prisms are placed at such equal angles above and below the horizontal as will fully correct the vertical, and partially the horizontal imbalance.

Prisms up to three degrees will usually be accepted placed in either way. They are tried out first as hook fronts over the regular glasses, later being ground in permanent form if their use affords relief from symptoms. In case they do not, the question of operation comes up if the deviation is great. If not, other causes of ocular symptoms are looked for, such as dental or intranasal conditions.

I am aware that these methods differ from those advised by most authorities. It is not rare to obtain relief from marked disability by prism correction of one degree of hyperphoria, therefore, in higher amounts, it seems unreasonable to leave that much and even more uncorrected. The almost constant relief from symptoms, except in cases where there is a large horizontal deviation or a marked convergence insufficiency, proves, to me at least, the merit of the method.

Operative Treatment.

In the presence of proof that the deviation found is the cause of the symptoms and eye disability, operation is indicated:

1. When prisms and lenses fail to give sufficient relief. Patients often are satisfied with relative comfort afforded by partial correction (case 4263, table I).

2. When, by operative correction of the deviation, the wearing of glasses can be avoided. This is an optional indication that is often taken advantage of by women. (Cases 2877, 4415, 4552, 4554, 4686, 4918, 5198, 6439, 6966, 7221, 7536, 716F.)

It is needless to emphasize the importance of finding the muscle at fault. Almost without exception, the diplopia tests in rotation, after occlusion, will show a lessening of the deviation on looking down and an increase on looking up. Therefore the superior rectus of the cataphoric eye practically always requires shortening. In some of my early cases (511, 524, 1346), I erred in following Worth's advice as to the difficulty and inadvisability of operating on the superior rectus. But for this error, Table II would show a clean sweep in favor of the superior rectus. Cases 3286 and 6378 are included in Table II to emphasize this argument; for in one a wide divergence, and in the other a visible convergence disappeared solely by shortening a paretic superior rectus. Case 3286 was shown at the 1923 A. M. A. meeting in San Francisco, and the condition is even better now than then.

Table II is made up of cases relatively low in degree, as I want to prove conclusively that accurate gradation of result is possible by my cinch shortening method.

When either occlusion or prism correction, or both, give full or marked relief, I feel justified in assuming that the cause of the symptoms has been found.

The patient must be fully informed as to the prospects before starting operative correction. If there is a multiple deviation, he must be told that, in all probability, there will be multiple operations. A perfectly successful correction of 10 degrees of hyperphoria will probably not fully relieve symptoms due to that plus 15 of exophoria, with possibly a weak convergence. I always tell my patients that, unless they will see the matter thru to what I think is the finish, I would prefer not to start. I explain why the restoring of the visual axes to parallelism is not always a one operation affair. As an unfinished result is no boost for the operator, and in order to encourage the patient to stick to the finish, I make a charge for the entire work no matter how many operations. I enjoy the work, and each operation takes only a few minutes. All are done in the office—even the children who require a general anesthetic. Tuesday and Thursday afternoons I regularly take off for golf, so I date my general anesthetic cases for those days at noon, finish about 12:30 or 1 o'clock and then skip out. The child stays till about four, when he is taken home, in a closed car, well bundled up. Here is the time and place to get in a boost for the advantages of the year round

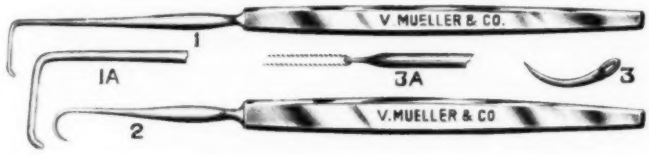
California climate. Local anesthetic cases I do at 9 a. m., before the routine office work. The only operations done in a hospital are the extraction stage of a cataract, and when a general anesthetic must be given to an adult. In this way I save an enormous amount of time for myself and money for my patients.

At the A. M. A. meeting in 1923, I demonstrated my method of shortening, with details of graduating the results to Dr. G. E. de Schweinitz and several other ophthalmologists. Dr. de Schweinitz asked me to give him a short description for the present edition of his book. In this paper, I wish chiefly to describe the method of graduating the result, and this can be done by describing the handling of case 6966 from start to finish.

No. 6966. Seen first on July 10, 1925. Marked eye disability. Headaches frequent and severe, especially so after trips in automobile. Symptoms definitely related to use of eyes. Wearing: R. E., + cyl. .50 ax. 30; L. E., + cyl. .25 ax. 150. My refraction under cycloplegia was the same in right eye, and plus sphere .50 in left. This was not enough difference to be worth while. Esophoria 1 and vertical orthophoria. So the left eye was patched. After four days, there was a vertical deviation of five degrees, with lateral orthophoria. There was immediate and complete relief from all troubles, while wearing the patch, even when in an auto. Prisms were given combined with her correction, and a few days later she started for her home in Montana by auto. On August 2nd, she wrote me as follows: "I had one headache on the long drive home, but by wearing the patch the next day for a while, I had no more trouble. With that exception, I have had no feeling of eye-strain whatever. I am very glad that I consulted you as I feel better than I have in five years."

She returned on Nov. 23, 1925 with the idea of having operation, as she wanted to avoid glasses if possible. (She is a very good looking girl.) In order to be certain that she could do this, she wore the patch and no correction for the right eye, with no discomfort, proving that the oblique cylinder in the right eye in itself was not causing much trouble. She then wore a pair of prisms, with no lenses in either eye, and was comfortable. Ten days were spent in these tests, which satisfied her. Under the patch, there was definite increase of the diplopia in looking up, the right eye lagging, indicating

that the right superior rectus was the muscle to shorten. On Dec. 4, 1925, that muscle was shortened, using four strands of Bauer and Black medium suture material and many narrow tendon bands. This gave an immediate overeffect of 6 degrees unchanged by removing one strand. She was told to take the patch off when at home, and practice ceiling to floor exercises. Dec. 5th, there was 9 degrees overeffect, so one more strand of shortener was removed and exercises continued. On the 7th the overeffect was down to 4, and by the 9th only ½ de-



gree. The patch was replaced and the next morning the overeffect was back, so patch was removed. On the 14th (ten days after operation), the test was ½ degree right hyperphoria, and at this time the remaining shorteners were removed. Between the 10th and 14th, she had used her eyes as she wished, going to the movies and taking auto rides, with entire freedom from discomfort. On April 23, 1926, she tested exactly the same, and reported "Not a bit of trouble." She used her lenses only for reading and the movies, and is a most appreciative patient.

Time or space would not permit a detailed description of the operative technic. This has been given most fully in the 1916 Transactions of the A. M. A., Ophthalmologic Section. I hope in the near future to prepare an article giving the present method of carrying out the original shortening principle. I think an actual demonstration of the shortening will be more enlightening than a long description.

TABLE I.

MEASUREMENTS AFTER OCCLUSION OF ONE HUNDRED TEN APPARENTLY ORTHOPHORIC CASES.

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
783	..	5	..	3	Entire relief.
1323	6	..	..	4	After 8 years' care, right internus shortened, giving relief.
1832	..	1	..	2	Great improvement.
1983	..	4	..	7	Definite paresis right superior rectus. Refused operation.
2313	5	2	..	3	Marked improvement.
2350	5	2	..	1	Not much relief. Is a microscopist.
2626	..	..	..	2	Entire relief. Is a draughtsman.
2872	..	8	..	2	"First real comfort in a long time."
2986	..	14	..	2	Operative correction of exophoria.
3079	10	4	..	2	Left internus shortened, after which "very much better."
3106	..	4	..	4	Entire relief.
3107	..	..	..	1	No relief from prisms. Persuaded finally to wear spectacles instead of eyeglasses, and discomfort lessened.
3456	5	5	..	2	"Reads as long as necessary without trouble."
3589	..	..	..	5	Much better.
3621	..	1	..	3	"No trouble even in prolonged work."
3639	..	2	..	1	"Severe brow and neck pains have disappeared."
3708	..	6	..	2	Entire relief from headache and neck pains.
3909	..	..	..	2	Slight relief only. Is neurasthenic and hyperthyroid.
3964	..	3	..	1	Improved somewhat.
3982	9	5	..	2	Not helped, probably because of poor convergence.
4024	6	..	..	3	Not helped, probably because of poor convergence.
4182	5	4	..	4	Entire relief from severe headaches.
4231	..	5	..	2	Not much help. A chronic invalid and neurasthenic.

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
4292	6	5	..	4	Able to read two hours without trouble.
4311	6	5	..	3	Entire relief. Severe continuous headache stopped immediately on starting occlusion.
4390	..	3	..	10	Operation refused.
4394	..	..	..	2	Did not return for treatment.
4412	..	4	..	3	Patch immediately relieved headaches, nausea and dizziness.
4415	..	8	..	5	"Works all day and reads all night without trouble," after shortening the right superior rectus.
4435	..	6	..	3	Entire relief from date of starting patch.
4483	6	..	..	1	Headaches, present as long as can remember, stopped from date of patching. Emmetropic.
4567	8	6	..	2	"Gets along all right."
4585	..	3	..	2	"Awful headaches" lessened to "not bad enough to worry about."
4649	..	.	..	1	"Eyes are fine and no trouble." Dozens of patients have come to me thru this one.
4684	..	1	..	2	"Severe continuous headache relieved."
4686	6	4	..	9	Right sup. rectus shortened. Has to wear prisms for reading help.
4748	..	4	..	2	Severe headaches attended with nausea and vomiting entirely relieved.
4821	..	4	..	2	Relief. Emmetropic.
4878	..	7	..	1	More comfortable with patch but did not finish.
4911	5	2	..	4	Did not finish.
4918	..	..	..	9	Rt. sup. rectus shortened. "Reads for hours and no trouble."
4949	..	2	..	2	No better. Much intranasal pathology and surgery.
4984	..	3	..	3	Entire relief from frowning, with deep brow wrinkles, altho only eleven years of age.

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
5017	..	6	..	2	"Reads with great ease."
5022	..	1	..	2	"Entire relief—can't go without correction."
5080	..	6	..	3	Great relief and marked lessening of a facial tic.
5098	..	3	..	2	"No more headaches or discomfort after study."
5178	..	2	..	1	Did not complete examination.
5195	..	..	1	3	"No trouble now in using eyes." Both eyes emmetropic.
5310	..	4	..	3	"Entire relief from headaches." Both eyes emmetropic.
5348	5	1	..	1	"No trouble in reading or from light."
5378	..	5	..	7	"Entire relief from headaches of many years duration."
5440	6	1	..	3	"Can't get along without prisms." Both eyes emmetropic.
5445	7	4	..	2	"Considerable relief."
5465	..	2	..	4	Not much relief.
5731	..	8	..	2	"Entire relief from headaches and all symptoms."
5845	6	4	..	4	"Entire relief."
6074	5	4	..	1	"Reads a long time and no trouble—feels fine."
6155	..	3	..	3	"Sews constantly and no trouble." This case emmetropic in each eye.
6286	..	5	..	3	No relief.
6348	..	1	..	1	But little help.
6394	..	2	..	1	No relief.
6395	6	3	..	2	"Entire relief and no more neck pain."
6437	..	1	..	6	An out of town patient. Reported for test 18 mos. after first examination. In this time, vision in one eye had failed—found to be due to retinal detachment.
6563	5	4	..	2	Not much relief. Had to have frontal sinus opened.
6669	..	6	..	1	Reads as long as four hours without trouble.

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
6688	..	4	..	3	Patch relieved symptoms—did not report effect of prisms.
6766	..	9	..	4	Comfort with patch. Did not report effect of prisms.
6891	5	1	..	2	Marked improvement.
7221	5	5	..	6	Right sup. rect. shortened, with entire relief. An optional case as refraction was practically emmetropic.
7367	..	2	..	2	No symptoms. A candidate for West Point who desired a full test of muscles.
7398	..	7	..	3	Patch relieved symptoms of 15 years standing. Prisms also, but preferred operation, which also gave relief. Emmetropic in both eyes.
7402	..	4	..	2	"No doubt of improvement with prisms."
7428	..	3	..	2	No trouble with near work. Practically emmetropic.
7495	5	6	..	2	"Can read and sew without eye aches." Emmetropic.
7536	..	6	..	9	Right sup. rect. shortened, with entire relief from headaches, carsickness, etc. Practically emmetropic.
7804	..	8	..	3	"No more headaches." Emmetropic.
7087	5	5	..	..	Comfortable with patch. Symptoms due to weak convergence. Did not complete treatment.
6727	5	..	6	..	"Great relief in all uses of eyes. No trouble in spite of prolonged reading."
5643	..	4	..	1	No help.
5319	..	8	..	..	Horizontal prisms failed to help. Referred for nasal work.
4401	16	3	..	1	After patching, there was no attempt to converge. Operation advised but refused. Emmetropic.
2875	5	9	..	..	Comfortable with patch. Operation advised but refused.
1068	..	..	2	..	No prisms ordered.

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
898	6	3	..	..	Comfortable with patch. Did not return.
6489	..	..	..	3	Entire relief.
4263	5	14	..	12	Prisms 3 degrees up and down gave great relief from headaches of many years duration. Operation was advised.
1298	..	..	..	2	"Great relief from lifelong disability."
2505	..	1	..	4	"Entire relief from headaches, dizziness and eye discomfort."
206	..	7	..	5	"Very much better."
3435	10	8	..	3	One internal rectus shortened, giving esophoria 1 with convergence to 3 inches. No symptomatic relief, as case was a bedridden neurasthenic of the worst type. Operation done at own request and that of family physician in hopes of obtaining relief.
2230	..	5	..	4	"Entire relief from all symptoms—never felt so good."
264	..	3	..	1	"Less uncomfortable."
469F	..	8	..	3	Entire relief. "First genuine comfort in years."
239F	..	6	..	5	"Better." Emmetropic.
499F	..	1	..	1	"Very much better and but little dizziness."
4049	..	6	..	2	No symptoms, being found while occluding one eye in treatment of corneal ulcer.
716F	..	11	..	5	Optional shortening of sup. rect. with entire relief in all uses except reading. Prisms 3, bases in, permitted reading till onset of presbyopia. Practically emmetropic.
742F	..	..	..	3	Entire relief. Practically emmetropic.
895F	..	6	..	6	"Feels fine with prisms."
988F	5	1	..	4	Entire relief from severe headaches and eye disability. Had been under the care of a Prof. of Ophth. in one

Case	Convergence (inches)	Exophoria	Esophoria	Hyperphoria	RESULTS OF TREATMENT
					of our universities. I could not change his lens correction.
993F	..	..	..	2	"Great relief."
997F	4	4	..	4	No relief from prisms. A neurasthenic woman with night and morning headaches. Refused cycloplegia.
521F	..	4	..	2	Entire relief.
828F	..	3	..	2	Lens correction without prisms gave relief.
056F	5	2	..	2	"Less uncomfortable."
629F	7	2	..	..	Entire relief with patch, which was used to prove weak convergence the cause of symptoms. Practically emmetropic, so operation was advised but refused.
696F	..	3	..	1	"Amazed at relief from dizziness and carsickness."
6565	..	4	..	4	Entire relief.

TABLE II. THIRTY-THREE SHORTENINGS OF THE VERTICAL RECTI.

	Before Operation					
CASE	Hyperphoria	Cataphoria	Exophoria	Esophoria	Rectus shortened	RESULT
291	R. 16	L. 12	8	..	L. Sup.	R. hyper. 2. My first operation by present method.
511	L. 7	R. 7	8	..	L. Inf.	Vert. ortho., exo. 8.
524	L. 5	R. 4	..	2	L. Inf.	Orthophoria.
1328	L. 17	R. 17	3	..	R. Sup.	L. Hyper. 6. This was done before working out method of graduating result.
1346	R. 12	L. 12	10	..	R. Inf.	Vert. ortho., exo. 4.
2553	L. 10	R. 10	3	..	R. Sup.	Vert. ortho., exo. 1

CASE	Before Operation: Hyperphoria	Before Operation: Cataphoria	Exophoria	Esophoria	Rectus shortened	RESULT
2597	R. 7	L. 5	..	..	L. Sup.	Orthophoria. This case orthophoric before patching.
2858	L. 10	R. 5	11	..	R. Sup.	Orthophoria.
2877	L. 6	R. 6	..	1	R. Sup.	Vert. ortho., eso. 3.
3110	L. 8	R. 8	5	..	R. Sup.	Orthophoria.
3286	R. 15	L. 15	..	..	L. Sup.	Parallel. Case of esotropia with marked upshoot of right eye on looking left and up.
3737	L. 12	R. 12	..	..	R. Sup.	Left hyper. 1.
4415	L. 5	R. 5	10	..	R. Sup.	Right hyper. 1/2, exophoria 3. Emmetropic.
4552	L. 4	R. 4	8	..	R. Sup.	Vert. ortho., exophoria 2. Practically emmetropic.
4554	L. 25	R. 18	1	..	R. Sup.	Left hyper. 1/2. Practically emmetropic.
4686	L. 9	R. 9	4	..	R. Sup.	Right hyper. 1/2. This case was orthophoric before patching, also emmetropic.
4751	R. 5	L. 3	2	..	L. Sup.	Orthophoria and no diplopia, which had been very annoying. L. sup. paresis.
4918	L. 9	R. 8	..	..	R. Sup.	Right hyper. 1/2. Emmetropic and orthophoric before patching.
5198	R. 6	L. 6	3	..	L. Sup.	Orthophoria. Emmetropic.
5909	L. 4	R. 5	10	..	R. Sup.	Orthophoria. Only an exo. 1 before patch.
6085	L. 10	R. 6	15	..	R. Sup.	Right hyper. 3, left cata. 1, exo. 4.

CASE	Before Operation: Hyperphoria	Before Operation: Cataphoria	Exophoria	Esophoria	Rectus shortened	RESULT
6439	L. 4	R. 2	13	..	R. Sup.	Right hyper. 1/2, exo. 3, with disappearance of definite diplopia on looking up.
6966	L. 5	R. 3	..	..	R. Sup.	Right hyper. 1/2. An optional operation.
7044	L. 6	R. 6	4	..	R. Sup.	Orthophoria.
7221	L. 6	R. 5	5	..	R. Sup.	Right hyper. 1/2. Emmetropic and orthophoric before patching.
7473	L. 8	R. 8	6	..	R. Sup.	Orthophoria.
7536	L. 9	R. 9	6	..	R. Sup.	Right hyper. 1/2. Orthorphoric before patch. No lens correction needed.
6378	L. 20	R. 7	20	..	R. Sup.	Orthophoria by Maddox rod and cover test in both vert. and horiz. directions.
716F	L. 5	R. 5	11	..	R. Sup.	Orthophoria. Orthophoric before patching. No lens correction needed.
791F	L. 7	R. 7	2	..	R. Sup.	Right hyper. 3. Prisms control.
639T	L. 16	R. 10	4	..	R. Sup.	Left hyper. 3, right cata. 3. Discomfort entirely relieved. Diplopia had troubled much.
1100F	L. 10	R. 10	4	..	R. Sup.	Vert. orthophoria, esophoria 1/2. Test before occlusion: R. hyper. 1/4, eso. 1/2.
8000	R. 5	L. 5	6	..	L. Sup.	Vert. orthophoria, exophoria 1.

DISCUSSION.

Dr. Walter B. Lancaster, Boston: I wish to compliment Dr. O'Connor on his careful study and his very good results. The latter is what count—good results. I cannot duplicate either the careful study or the results. I have used occlusion for a great many years, but I do not use it as much as he does. I rarely use it for diagnosis. I use it not infrequently for treatment. If I have a case with severe symptoms and inability to use the eyes, where there is a large deviation, too large to correct by prisms, and the question of operation comes up, I frequently cover one eye for a period. The relief from this treatment usually is a strong argument with the patient to submit to operation. They get tired of using the cover. On the other hand, I have a patient who is an elderly professor in a college. For some years, she has been doing work in her room and in the library with a patch on one eye, and going about and doing class work and so forth with both eyes uncovered. She gets so perfect satisfaction in this way, that I do not urge her to have an operation.

I think Dr. O'Connor ought to accumulate, for our benefit, a series of 100 to 200 cases of occlusion of the eye in normal cases, patients without asthenopic symtoms. I suspect that the amount of deviation found by occlusion has not the significance that he attaches to it. I should certainly hesitate in a patient who showed 13 prism diopters of hyperphoria after a week's occlusion, altho none was manifest before—I should hesitate to operate to correct that defect, fearing lest I should produce an error in the opposite direction. But Dr. O'Connor can perhaps report actual cases where he has done that very thing, and show us that there is nothing to be feared.

I wish to emphasize this point in regard to diagnosis, that it is rare that any condition which comes to us is due to one thing alone; it is rare that headaches are due to the eyes alone, and it is rare, when they are due to the eyes, that they are due to ocular muscles alone. Usually there are several factors at work, and if we remove one or two, Nature takes care of the others. If I were to rewrite the first paragraph of Dr. O'Connor's abstract, instead of saying: "A hidden vertical deviation is the cause of a persistence of asthenopic symptoms after accurate correction of a refractive error and any apparent heterophoria," I should say: "The commonest cause of persistence of asthenopic symptoms after careful correction of refractive error is the failure to correct fully the hypermetropia." As I see cases which have been treated by myself and others without relief, and then reexamine them and do give them relief, it is most frequently by correction of the refraction in a more satisfactory manner, and not by correction of muscular defects. In other words, muscular defects are important, but are less important than errors of refraction.

I am glad that Dr. O'Connor emphasized the importance of the spasmodic elements. I think that the lack of coordination is a matter of the nervous control of the muscle. Accommodation is a most important factor, one which causes esophoria by the excess, and exophoria by its lack. Also, when the patient attempts to correct a vertical deviation, this

causes spasms of various muscles. Here is one more reason for emphasizing the fact that the phoria is not due to some weak muscle or to some anatomic defect; it is due to defective nervous coordination. This is what makes it so complicated and so difficult for us to agree upon. Dr. O'Connor has made an important contribution for our study.

Dr. Frederick Stauffer, Salt Lake City: There are two phases of this subject that were not touched upon by the essayist, which I wish to bring to your attention. First, I would like to ask him if he noticed any influence of the disease of the ethmoid cells or the sphenoid sinuses anywhere in the nose upon these hyperphorias. And second, I would like to know how many of these cases of hyperphoria recover without any operation after the use of prisms for a considerable period of time. I have a number of case records where as high as six degrees of hyperphoria has become entirely normal after wearing prisms for three or four years. I found the symptoms of eyestrain were promptly relieved after giving the prisms, and then a test in two or three years would show that they required a lessened degree of prisms, and after a while I have had to remove the prisms entirely in some cases. Of late years, I have noticed that a great many of these hyperphorias are due to conditions in the nose, a pressure causing reflex disturbance. I think a great many temporary hyperphorias are due to disease of the nose, and if the nasal conditions are corrected, you will relieve the hyperphoria.

Dr. E. N. Robertson, Concordia, Kans.: As to the most common cause of these eyestrain symptoms, I suppose we can argue both ways and have a good deal of evidence on both sides, but I wish to bring argument in favor of what Dr. O'Connor has said in that respect. I have had a number of cases that I have rechecked pretty carefully with the assistance of very able men, and we found a vertical deviation of 2 to 4 degrees which was not corrected by a prism, but in every case relief was given to the patient by using practically the same lens as before and incorporating the correction for vertical deviation. I think we should be very careful as oculists in testing for these deviations, because it is my observation that very few optometrists will pay attention to such things.

I wish to mention a case that came to me about six months ago, a little boy, seven years of age, who the mother said for several years, in fact, ever since he began to walk, had a turning of his head to one side. When he wanted to look at anything particularly, he always closed one eye. He had a great deal of headache and it always made him sick to ride in an auto or on the train. He had between 8 and 9 degrees of hyperphoria, which we corrected by prisms, and he got relief from headache and other symptoms.

Dr. Joseph L. McCool, Portland, Ore: Too much emphasis cannot be placed upon the subject of the relationship between vertical and lateral imbalance. A careful study of many of our cases of esophoria and exophoria will frequently reveal an underlying vertical insufficiency. I believe this to be true to a great degree in cases of convergence insufficiency. A careful study of the diplopia field will often unmask a vertical error.

I have been using the occlusion method for four or five years, but lacking the persuasiveness of Dr. O'Connor, I have never been able to induce my patients to wear a patch for more than twenty-four hours. Women, particularly, balk at this procedure. I frequently have the patient wear one in the office for an hour, or have one worn over night. I am sure that this method is valuable, for in many instances it has revealed a hyperphoria that was unsuspected, or has increased an already existing one. We must assume, of course, that the cases the essayist speaks of are intrinsic hyperphorias. In this connection it is well to remember, that in individuals past middle life, hyperphoria may be the result of transient paresis of the ocular muscles resulting from tabes and general paresis.

Do not overlook the value of the parallax test in diagnosing many of these insufficiencies. I find it a very valuable test—far superior to the Maddox rod.

O'Connor's operation, which I have been using for five years, is of particular value in convergent insufficiencies. It is in these cases especially, that you not only want the effect, but you want to be sure not to leave your patient in a worse plight than before you operated. I would be loathe to use a resection or advancement operation in these cases, because it is difficult to do. My experience with O'Connor's operation in these cases has been very satisfactory, and since he has changed from the use of catgut to many strands of fine or medium dermal suture, it is capable of very accurate adjustment. As a matter of interest, I have found, by actual measurements in some of my squint cases, that 12 strands of medium dermal suture will shorten the tendon and muscle 12 millimeters.

There is one criticism of Dr. O'Connor's paper that I should like to make. I am inclined to believe that a vertical deviation of from 15 to 25 degrees would be a hypertropia rather than a hyperphoria.

Dr. Luther C. Peter, Philadelphia: I want to endorse one point in Dr. O'Connor's paper particularly, and that is the full correction of hyperphoria. Most of the text books and teachers have laid down the dictum that we should correct about two-thirds. In my experience, results are much better when I give a full correction, after I have satisfied myself that the patient has a definite and stable amount of hyperphoria. Sometimes there will be a difference between the far and near correction, and in those instances I usually prescribe the minimum correction, unless there is a great difference in the amount of hyperphoria for far and near.

Like one of the former discussers, I would hesitate to operate upon a case of ten or twelve degrees of hyperphoria which was uncovered only after occlusion for eight days. I would rather attempt to correct as much as possible by the use of prisms, and observe whether this degree of hyperphoria would be maintained.

One of the discussers spoke of the influence of the diseases of the ethmoids and sphenoids, and I am glad he mentioned it, because in those cases in which one sometimes finds it necessary to put on prisms of three or four diopters, and then in two or three years finds the patient overcorrected, I think we will find in the majority of instances the hyperphoria is caused by ethmoidal and sphenoidal disease.

Dr. William H. Crisp, Denver: I want to emphasize some of the points made by Dr. Lancaster. I am particularly interested to hear a veteran in this work, one who has done so much original work in refraction, speak of the fact that any of us may at times find a possibility of improvement in our refraction work. I think I am a very careful refractionist, and yet more and more I feel that there is never an end to the possibility of improvement in difficult refractive corrections. Any patient may show appreciable change in the course of a year or two, but where I have gone over a case recently and the patient still complains, I may find something wrong with my previous measurement. In such a case, I do not blame myself or the patient, but attribute the trouble to special difficulties, some irregularity of focus with regard to which, after the eyes have worn the correction for a while, further improvement can be made. I do not believe there is any refractionist in the world who may not have this experience.

I should be loath to believe that so large a proportion of our patients have hyperphoria which is responsible for particular symptoms. I have had some experience, personally, in this line as a patient. I test the muscles rather carefully in practically every one of my patients. I have observed several times, that apparently well defined vertical muscle errors completely disappeared after revision of the refractive correction. It is necessary to remember, that in refraction above every thing else we ought to "be not weary in well doing."

Dr. Roderic O'Connor, Oakland, Calif., (closing): In regard to the question of accurate refraction, I think Dr. Lancaster has forgotten the emphasis I laid on the care taken in this respect, to eliminate, as far as possible, the possibility of error. As stated in the body of the paper, many cases were emmetropic. Many were perfectly comfortable with prisms added to a correction known to be wrong, or used alone without a correction for a definite known refractive error, showing that, in many cases, the muscular imbalance is the cause of greater symptoms. This was the case in all those I called "optional operative," in which especial care was taken to be certain that the hyperphoria was the sole cause of the symptoms.

Occlusion is of benefit, also, in causing apparent deviations to disappear, thus proving that a certain number are false. Recently, in this way I saved a man from operation, advised by another ophthalmologist, for 8 degrees of esophoria, which under patch and accurate refractive correction reduced to 2 degrees, with corresponding relief from symptoms.

As mentioned in the paper, the intranasal apparatus is always investigated carefully when correction of ocular defects fails to relieve symptoms, with frequent success. However, I do not believe that intranasal abnormalities can cause a true muscular imbalance.

In regard to the question of safety of correction based on patch test: I have hundreds of cases running as far back as 1919. Those who have returned at intervals still take approximately the same prism correction —they hold straight with continued relief from symptoms.

In other words, I take all possible precautions in order to insure symptomatic as well as mechanical results. Only in this way can I expect satisfied patients who, after all, furnish most of any man's practice.

Those who refuse to see the importance of this work are like the ostrich who pokes his head in the sand to hide from danger.

GASTROINTESTINAL TOXEMIA AND ITS RELATION TO OCULAR PATHOLOGY.

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A study of this subject leads one into many fields, but eventually narrows down to a few major points; first, the theory of gastrointestinal toxemia; second, the causes of gastrointestinal toxemia; third, its mode of entry into the system and its effects on the body as a whole; fourth, its effect after entry on special organs, in particular the eye.

The idea of the absorption of intestinal enzymatic food and putrefactive substances as an effect of intestinal stasis, has been accepted by the profession for so many years, that one almost needs courage to argue against it. The supposed clinical picture is an old, familiar one—constipation, headache of varying degree, coated tongue, bad breath, malaise, backache and a host of other symptoms are included. The fact that these general symptoms are relieved promptly and efficiently by various measures such as cathartics, cholagogues, enemas, surgical measures and what not, would seem to dispel all doubts as to the correctness of the gastrointestinal toxemia theory. The literature in support of it is voluminous. Gradually such terms as autotoxemia, autointoxication and others have been used loosely and interchangeably, until the whole subject has become a jumble. Writers will refer to a toxemia, implying an intestinal origin, when they are really talking about some change in the blood chemistry. What one person will refer to as an intestinal intoxication will be a protein sensitization, etc. Many writers who have believed in the truth of the intestinal toxemia theory, have nevertheless realized in late years that more proof was necessary to establish it on a strong foundation, and the whole question is again opened up. A more scientific study is being made to demonstrate the soundness of their views. This study has been stimulated by the work of advocates who take the opposite view of the question, and attempt to prove that the gastrointestinal tract is not a source of origin of somatic disease.

I will pass over the older literature, from which volumes might be produced citing cases of diseases of all kinds, including the eye, which have been apparently cured by enemas, cathartics, and other methods of cleaning out the bowels. This empirical method of establishing a fact is no longer accepted in scientific circles as adequate proof. Such information can be derived from the work of the older authors, Bouchard, Combe, Metchnikoff, Lane and many others. Among the more recent writers, James Walker reports in the Lancet, 1924, three cases of functional mental disorders due to toxemia from the bowel. While they are fully worked out, they are not convincing as an argument in favor of the theory under discussion.

One of the best known authors on intestinal stasis, Lane, who has been investigating its causes and results for many years, has recently had published an article in the British Medical Journal, 1924. He mentions "changes brought about by the absorption from the infected contents of the gastro-intestinal tract of more toxins than the liver is able to take care of. These noxious substances getting into the circulation are carried to every organ and tissue in the body, and produce disastrous results in proportion to the toxicity of the blood." "Besides the changes that are the direct result of the supply of toxic blood to the tissues, we have a number of infections by organisms, which occur because of the inability of the degenerated tissues to withstand them."

As you will note, Lane states these as facts, and we presume that he would not make these statements if he had not demonstrated them or had others do it for him. Lane has devoted a great part of his professional life to the subject of intestinal stasis and its evils, and his views are entitled to our serious consideration.

I quote another writer, Eustis Allen, in the Southern Medical Journal, in an article January, 1924. He states that few of our foremost internists, both in the North and in the South, consider the question at all, and the routine examination of the urine for indican is neglected in most of our best hospitals.

Mizele demonstrated, that when the intestinal flora was transformed from the putrefactive to the acidophilic type, a high percentage of his cases were relieved of symptoms often ascribed to neurasthenia. It has also been shown by Bass,

Rettger and others, that the intestinal putrefactive bacteria can be replaced by feeding cultures of the bacillus acidophilus.

Herter and his associates, Dale and Laidlow, Ackerman, Kutsch and others have demonstrated that a number of the amins, the result of protein putrefactive changes in the intestine, possess definite physiologic action. If this is true, it can readily be believed that the eye and other organs are subject to various pathologic changes as a result. He enters a plea "for recognition of intestinal toxemia in the treatment of definite pathologic conditions in which absorption of poisons from the intestinal canal may influence the symptomatology and even the prognosis of the disease," and concludes: (1) "that laboratory and clinical evidence is sufficient to warrant a recognition of the existence of intestinal toxemia, and (2) consideration should be given to the presence of intestinal toxemia in the treatment of all patients, and careful statistics tabulated in order to determine its true importance."

Enough has been presented, I think, to show that a large part of the profession is committed to a belief in the gastrointestinal theory of absorption of putrefactive products. I am now going to quote largely from an exhaustive article by Alvarez in the Phys. Review, Vol. 4, for July, 1924. In it he attempts to combat many of our preconceived notions of gastrointestinal toxemia. There are 32 pages of interesting facts, and the article is so comprehensive, that it would seem superfluous to go over the same mass of literature which he has drawn on for presenting his case, even tho he concedes that the question of absorption of toxins is still open in many respects. He very clearly lays down premises which must be proven before the theory can be accepted, as follows: "It is not sufficient simply to show that toxic substances can be found during the enzymatic and bacterial destruction of foods. It must be shown, also, that these processes take place in the digestive tract, that the substances are found in sufficient amounts, so that they can give rise to either acute or chronic poisoning; it must be shown that they can pass thru the mucous membrane, normal or diseased; and it must be shown that they can escape destruction in the liver.

"It must next be demonstrated that the repeated injection of small doses of these substances into animals or into man will produce symptoms or pathologic changes similar to those supposedly produced by constipation in man."

He points out that very little exact work along these lines has been done. He enumerates a number of substances that are harmless or inert when taken into the gastrointestinal tract, which act profoundly on the organism when introduced into the circulation, the inference being that the same thing might be true of the toxic substances under discussion, and quotes numerous experimenters who have demonstrated this to be a fact. Among these substances are diphtheria antitoxin, tetanus toxin, insulin. Large doses of tuberculin given to calves by mouth produced no reaction. Large doses of salvarsan per rectum produce very little effect. He points out that if the gastrointestinal toxemia theory were true, the symptoms that go along with the constipation would not be relieved by emptying the bowel. He further presses this point home, by insisting that these symptoms are due to mechanical pressure on the rectum by the fecal masses. He quotes Donaldson and Pape, who produced all the classical symptoms of "autointoxication" by simply packing the rectum with cotton. In addition, there is the common observation that people may go indefinitely, you might say, without a bowel movement, and feel practically well. I quote one such case referred to in Alvarez's article. A man, during a period of 18 years, went from three weeks to three months without a bowel movement. At the age of 29, he went for five and, later, six months without a bowel movement. Finally he went from June 18, 1900 to June 21, 1901 without a movement. During these periods of constipation, he ate well and did a good day's work. He had gas and some pain and distention. He then passed about eight gallons of feces, lost weight and became weak. After the colon was cleaned out, he rapidly recovered. Out of all this controversy, one must conclude that there are two factors to be considered: (a) those from products of putrefaction and fermentation in the bowel, and (b) those from the bacteria in the bowel.

Those who contend that the products of normal digestion of proteins, carbohydrates and fats taken in abnormal quantities are a source of bodily disease, have not advanced enough proof to warrant their acceptance. It has been proven that the protein putrefactive compounds pass thru the intestinal mucosa, but they are, in most instances, taken care of by the liver. The chief compounds of protein digestion, the amino acids, have not been entirely proven such a source of disease;

the same thing is true of the products of carbohydrate digestion. The fatty acids and other substance can be definitely ascribed as a cause of disease changes in the intestine itself, but not so definitely in other organs and tissues of the body.

Passing to the products of putrefactive action in the bowel, we find the most important and widely studied is indol, and also its derivative, indican. Investigation of its formation and action has been going on for fifty years or more. It has been shown to be present in and significant of constipation and its syndromes. On the other hand, cases of long standing and obstinate constipation have been observed with little or no indican in the urine. Experiments with indican have been carried on in amounts much larger than those found in the intestines. Herter concludes that it is the only aromatic product absorbed in large enough quantities to have a toxic effect. Wells feels that the amounts of indican found in the urine ordinarily are so small in proportion to the doses used for experimental purposes, that it is doubtful whether this substance has any practical importance. The other putrefactive products, more especially indol, acetic acid, phenols, skatol and histamin, have been proven to pass thru the bowel, but in such small quantities, as to be of no importance as a causative factor of disease.

Of important interest is the subject of the passage of live bacteria from the bowel into the lymphatic glands and blood. Adami and his workers have demonstrated this beyond a reasonable doubt, and here we have another plausible explanation of the intestine as a factor in the connection of disease in other parts of the body. While it is true that the greater number of bacteria seem to be destroyed before doing any harm, many of them are carried to other parts and organs of the body, where they may be a source of disease. An organ, such as the eye for instance, may resist bacterial action until it is injured or damaged in some way, when it becomes the seat of active infection. Adami feels that this action of bacteria from the intestine and not autointoxication is the explanation of intestinal stasis as a source of disease. He further states, "The term gastrointestinal autointoxication is pernicious and not to be employed by any self respecting member of our profession, save for so limited a set of conditions, that for ordinary purposes it may safely be expunged from our medical vocabulary."

It would appear from what has been presented so far, that so far as the eye is concerned, the gastrointestinal tract gives rise to two possible causes in its resultant pathology—the poisons resulting from putrefactive processes in the gut and the living bacteria. Both have been proven to pass thru the intestinal wall, into the lymph glands and blood stream. The amount of the two factors is small, as they are broken down or destroyed usually before they go far into the circulation. It appears to me as probable, that a small amount of bacterial infection is a more potential possibility as a cause of eye pathology than a small amount of indol, fatty acids or other putrefactive products, circulating in the blood.

Adami asserts that an organ or tissue frequently is the seat of a retrograde process, due to the actual presence of bacteria of low virulence. Rosenow has demonstrated the same thing. Such an organ or tissue is required continually to destroy small numbers of such bacteria, until a chronic or acute infection develops in the worn out or damaged organ. When we know that 46% of the nitrogen content of the bowel is supplied by bacteria of all kinds, we can be easily convinced that these bacteria might be a factor in the pathology of the eye, just as well as the putrefactive compounds. It is well to bear this in mind in treating ocular diseases by purging and such measures, as it has been shown that purging not only does not produce any lasting effect on the bacterial condition, but, on account of the increased fluidity of the bowel contents, the bacterial count is actually increased. Also, it is difficult to concede any permanent effect from purgation on the elimination of such putrefactive products as indol or indican and like toxic products.

While other writers in ophthalmology for a good many years have spoken of gastrointestinal intoxication as a source of ocular disease, the literature is not overburdened with actual experimental effort; on the other hand, much has been taken for granted. Ramsay, in his recent book, speaks of the indistinctness of vision accompanying functional derangements of the stomach and gives, as the undoubted cause, defective elimination of waste products. Under iritis he says, "The urine should be examined for indican." Under "serpigenous ulcer" he gives, under treatment, calomel and a saline purge, and notes frequently an improvement in the eye.

De Schweinitz quotes J. F. Shoemaker, and says chronic conjunctival congestion has been attributed to gastrointestinal

autointoxication. No proof is advanced to enlighten us on this point. Under keratitis profunda he says, "it is *probably* an expression of gastrointestinal autointoxication in some cases." Fuchs refers to the foci found in the teeth, tonsils, sinuses and intestinal tract as a causal factor in eye diseases. He does not state whether the intestinal focus is bacterial or putrefactive in origin.

Bell cites a case by Deutschmann of sugar found in the lens, aqueous and vitreous of a diabetic and says, "If the sugar poison can invade the aqueous, the vitreous and nutritive stream of the lens, then why can it not produce an inflammatory process in any tissue of the eyes?" What does he mean by sugar poison in reference to the sugar found in the lens? It is only necessary in this connection to point out that the sugar, so mentioned, is the result of a possible pancreatic disturbance, and does not properly come under the head of intestinal toxemia, and can not be eliminated by measures directed to clearing out the intestinal tract. Dr. Bell stresses the baneful effects of sugar in the diet and lays the bad effect to the various ultimate fermentative products in the bowel. His stand up to this point seems largely supported by physiologists. That these toxins pass thru the bowel in large enough quantities in the ordinary individual is, however, disputed by many authorities, and no definite proof has come to my notice that the byproducts of sugar fermentation in the bowel are a direct cause of ocular pathology.

Herter says the volatile fatty acids derived from the carbohydrates are irritants, and they might produce some disturbance in a person with an irritable stomach or small intestine. Alvarez quotes Dixon, supported by Porter, Meyer and Morris, "that the excessive formation of lactic acid in the bowel can rob the body of some of its bases and thus increase the normal excretion of calcium four or five times. Such calcium starvation would account for the backwardness of growing children." It is this class of children that we usually find are fed excessive quantities of sweets, and in whom we find such affections as eczematous keratoconjunctivitis, phlyctenular keratitis and such allied conditions. It is this class of children, also, who are most vulnerable to bacteria. These children are the victims, then, of nutritional disturbance, and not of a gastrointestinal toxemia. Is it not as reasonable to suppose that the bacteria found in the intestines find here a fertile

field in an irritable bowel, pass into the blood stream, and become the exciting cause of various inflammations of the eye?

Bell, in an article on intestinal toxemia, points out that every case of retrobulbar neuritis should have a clinical analysis of the blood to determine the amount of blood sugar, it being of value in the treatment. The blood sugar, as has been pointed out before, is not necessarily an indication of intestinal toxemia, as a large percent of the blood sugar may be derived from protein metabolism, whether the persons eats any sugar or not.

I do not wish to give the impression that I question Bell's stand on the proper supervision of the diet in the treatment of disease, especially in regard to the excessive amount of sugar. I am only questioning some of his conclusions as to the manner in which their ultimate products in the bowel effect the organism. I am heartily in favor of his sugar platform. In one respect, especially, I commend his ideas. He starts at the top of the intestinal tract instead of the bottom in his efforts to eliminate the cause of ocular disease in so far as the intestinal contents are a cause.

Last year, Dr. Weeks presented an article at Kansas City, which included this topic. He cites six cases of eye disease, including iritis, conjunctivitis, marginal keratitis and lens precipitates due to iritis. Two of these cases which he classes as due to intestinal toxemia were cases of ptomaine poisoning, and should not be included in the group. The other cases were the result of overeating, some of them including the overindulgence in alcohol and tobacco, which may or may not have been a factor in the etiology.

Conclusions.

1. Ocular diseases may be caused by the absorption into the blood of substances from the intestines which are derived from at least two sources, fermentative products as a result of digestion, and bacteria.

2. In view of the fact that the physiologists and research workers in various lines have not definitely come to conclusions as to how and to what extent these substances act as a cause, we, as oculists, should not commit ourselves to definite conclusions on these points, especially in the matter of treatment.

3. Chronic constipation, even with its presence of indican in the urine in appreciable amounts, is not a reliable sign in determining gastrointestinal toxemia as a cause of ocular disease.

4. Reports of cases of ocular disease which have improved or even been apparently cured by laxatives, cathartics, purges, etc., should not be accepted as proof by us that the toxic products of gastrointestinal digestion are a cause of ocular pathology.

5. We should try to determine, if possible, whether the condition of the patient we are studying is of gastrointestinal origin, whether it is due to changes produced in the liver and other organs, whether it is an allergy or sensitization, or as might be the case, due to an intestinal parasite. Botulism must also be considered.

Finally treatment should be along the line of proper dietary measures, those which will produce a minimum of putrefactive compounds and the establishment of conditions promoting the activity of the normal intestinal flora, as for instance in the use of the bacillus acidophilus, or any other agents having a similar effect.

DISCUSSION.

Dr. H. B. Young, Burlington, Iowa: This child, for which I was accoucheur in 1898, seems to have grown apace. I wonder sometimes, when I hear these discussions, whether I was wise in not obeying the injunction not to start something I could not finish. But I wish to call attention to this particular fact—that a great many things have been added to the subject which I did not contemplate. My original paper on this subject was on autointoxication amblyopia, and my cases were entirely defective vision without any structural changes in the eye—no inflammatory processes. Those of you who will look thru the proceedings of the American Medical Association for that year, will see that my most striking case was a woman who was totally blind, not even light perception, for three days without any eye pain whatever, as the result of improper feeding and the lack of entire quiet which was necessary after confinement. I think if we will stick entirely to those changes in the eye which are not structural, we will still be able to use the term "autointoxication amblyopia." I, myself, doubt whether it is proper to use it in those particular changes of the fundus and conjunctiva and other serious infections which, for lack of a better word, we say are autointoxication. I think if we hold to the original idea that I suggested at that time, the expression will be entirely proper.

Dr. Charles A. Bahn, New Orleans: Our conceptions of ocular pathology as related to primary disease of the gastrointestinal tract, are probably like the story of Robinson Crusoe—about five percent fact and

ninety-five percent fancy. We do not really know whether or not any of the normal or abnormal flora in the intestinal tract can produce metastatic inflammation in the eye. Nor do we know whether uveal disease is produced by actual bacterial metastatis or by soluble chemicals circulating in the blood and lymph. Unfortunately, we cannot usually determine, by the objective ocular findings alone, the source or type of the primary cause.

In the transmission of secondary ocular inflammation there must be three dominant factors: The causative organism or chemical, the sensitive individual and the sensitive eye, none of which are definitely understood.

Our treatment is, too, a rather hit and miss affair, based on the removal of all possible sources of disease, rather than on the removal of the one definite and specific cause. In many instances, we do not definitely know the primary cause, and often go about removing possible causes when the eye is sick unto death—when it is too late.

I, for one, feel indebted to Dr. Teale for having placed this interesting and important problem in the sunlight for frank and free discussion. In medicine, the sooner we really separate fact and fancy, the sooner will we solve our practical problems.

Dr. Frederick F. Teal, Lincoln, Neb. (closing): Dr. Young brought out one point, and that is that there is not much to the idea of diseases, aside from functional troubles, that can be ascribed to intestinal toxemia. Weeks, at a recent meeting at Kansas City, gave an instructive article in which he quote six or eight cases of specific ocular disease due to intestinal toxemia, but he made the same error I pointed out in this paper—two of the cases were due to ptomain poisoning, which properly speaking were not cases of intestinal toxemia. The poison of ptomain is found outside of the bowel and has nothing to do with intestinal toxemia.

THE REDUCTION OF GLAUCOMATOUS TENSION BY DEEP IRIDECTOMY.

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JOLIET, ILLINOIS.

That it is desirable to remove a portion of the iris root in iridectomy for glaucoma, has been recognized since the time of von Graefe.[1]

That this result is not easy of attainment, and is in many cases impossible, is shown in the eyes which reach the laboratories. Dr. Francis Lane,[2] pathologist at the Illinois Charitable Eye and Ear Infirmary, doubts the possibility of this ever being attainable.

The comparative thickness of the cornea, the smallness of the iris angle, the truncated cone shaped position of the iris as it rests in the aqueous, and the fact that the overhanging opaque sclera conceals the apex of the angle (the chamber bay) from the vision of the operator, all these are important anatomic factors which render any near approach to the ciliary border of the iris possible only when some technic is adopted which takes these factors into special consideration.

Then, too, as some of these natural obstacles are further increased by the pathologic state, it becomes more and more difficult of accomplishment.

In ophthalmic literature, there can be found recorded descriptions of over thirty operations designed to reduce tension, either by promoting internal drainage, as iridectomy, iridotomy and cyclodialysis, or external drainage, or combinations of both, as the Lagrange and Elliot methods, with their various modifications.

The writer is not adding to this list in the presentation of this paper, but acknowledges indebtedness to others, especially Török,[3] whose technic has been followed, except perhaps for the incision, more or less consistently during the past three years and in the sixty-seven cases herewith reported.

During this time, illustrative cases have been shown and the subject discussed before the Chicago Ophthalmological

Society,[4] and last year before this Academy.[5] Another year's experience and some experimental work, it is hoped, may justify a more thoro presentation of the subject.

Description of the Operation.

The operation is performed as follows: After proper preparation of the patient and the field, anesthesia is secured by instillations of a solution of 10% cocain and adrenalin, the last few drops being placed upon the iris after the incision is made. In highly congestive cases, general anesthesia is advisable. It

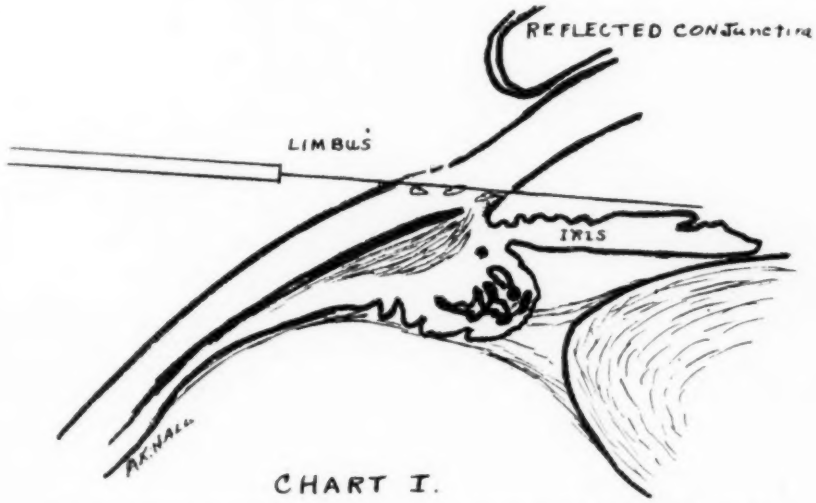

Fig. 1. Suker's drawing. Doubtful if any opening into the anterior chamber can be peripheral enough at site of puncture without injury to the iris.

is better also to have the pupil contracted, if possible, with eserin.

The globe is immobilized with the double fixation forceps of Ewing. The puncture is made above and to the outer side, in the sclera, as near as possible to the anterior surface of the iris. A cataract knife is used with a long tapering point, the Knapp and Smith knife. When the point can be seen in the anterior chamber it is pushed upward, so that, if possible, the extreme apex of the iris angle is located and the counter puncture made, forming an incision six or eight mm. long. The blade should not be turned outward too soon. The tendency in the cautious operator will be to turn away from the

ciliary body. This does not matter if the incision has been completed thru the sclera, so that the inner wound margin is in the apex of the iris angle. A rather large conjunctival flap is made with this incision.

By holding the corneoscleral-conjunctival flap away from the globe, the iris and ciliary body may be more or less separated from the sclera with the spatula. Then the iris is drawn

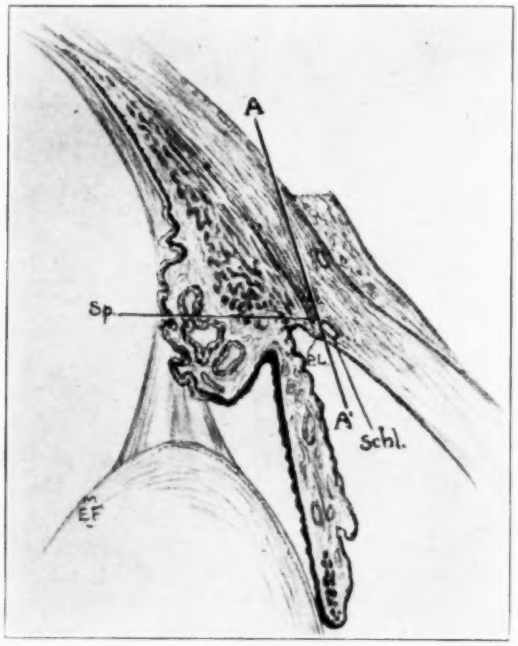

Fig. 2. Reese's drawing. Inner or chamber line of incision can not be made peripheral enough with keratome.

outward and downward with the forceps of Foster, and excised with the scissors of De Wecker or Barraquer. As will be shown later, it may be quite desirable to remove only a relatively small section of the iris, including, of course, the ciliary border, leaving one-half or three-quarters of the iris width between the operative coloboma and the pupil. The conjunctival flap is replaced without sutures. The best possible illumination, preferably daylight, and magnification with binocular loupe are important aids.

Fig. 3. P and P, possible lines of puncture. CP, line of counterpuncture —can be made peripheral.

COMMENT.

In the opinion of the writer, not enough attention has been paid to the incision.

Someone has said that the incision is the factor largely determining the success or failure of the cataract operation. For a different reason, this is true, also, in the operation under discussion. Col. Smith has shown how, in iridectomy, only that portion of the iris is removed which lies between the inner border of the incision and the pupillary border. In other words the iris peripheral to the incision is not removed. In entering the globe from without, it is probably not possible to reach the apex of the iris angle without injury to the iris. Reese[6] uses the keratome, entering the sclera 2 mm. back of the limbus. The external line of incision may be far enough back, but, on account of the angle at which it must be completed to

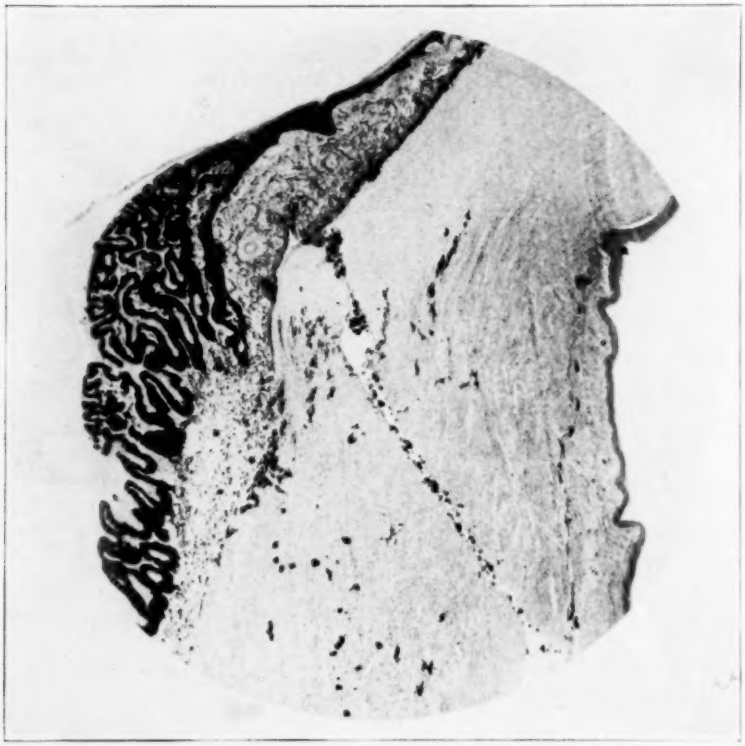

Fig. 4. Incision in pig's eye, not quite back in extreme angle.

avoid the iris, the inner or chamber margin of the incision is too far forward.

The same comment may be made in the technic of Suker.[7] While he uses the cataract knife, he uses it as the keratome is used, making no counterpuncture, therefore not reaching the iris angle. However, these two operators also depend on external drainage for the reduction of tension.

Török makes the puncture and counterpuncture 2 mm. back of the limbus, comparing it to the incision of Lagrange, but Lagrange makes puncture and counterpuncture only 1 mm. back of the limbus. He also depends on external drainage.

Treacher Collins[8] says that he "found the corneoscleral incision was always oblique, so that the external wound afforded only an imperfect criterion of the position and extent of the internal wound."

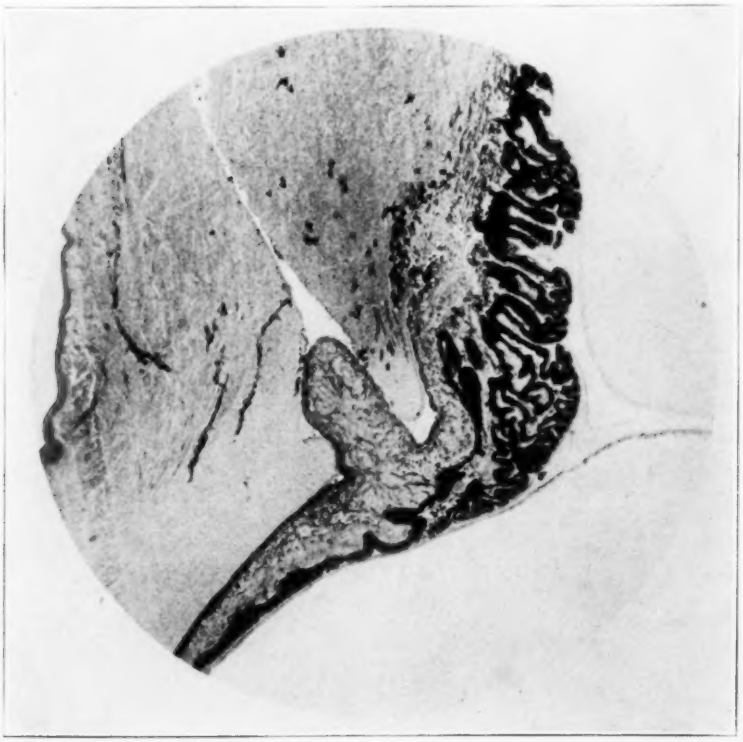

Fig. 5. Pig's eye incision, not quite in extreme angle; slight iris prolapse.

In the above description, it can readily be seen that one end of the incision will be farther from the limbus than the other, perhaps one or two mm., the end made by the counterpuncture being, of course, nearer the ciliary body.

It is just this counterpuncture over the extreme iridoscleral angle that insures the correct placement of the incision.

In the cases where the angle is not obliterated by iris, and the incision is properly made, the use of the spatula is perhaps not necessary. Where the iris is adhered to the sclera and the incision reaches only the apex of this false angle, then the iridocyclodialysis is necessary.

The fear of damage to the ciliary body by the extreme peripheric incision is not justified.

Verhoff[9] has performed cyclectomy, an operation in which he removes a portion of pars planum of the ciliary body.

The cases presented show that the forms of glaucoma

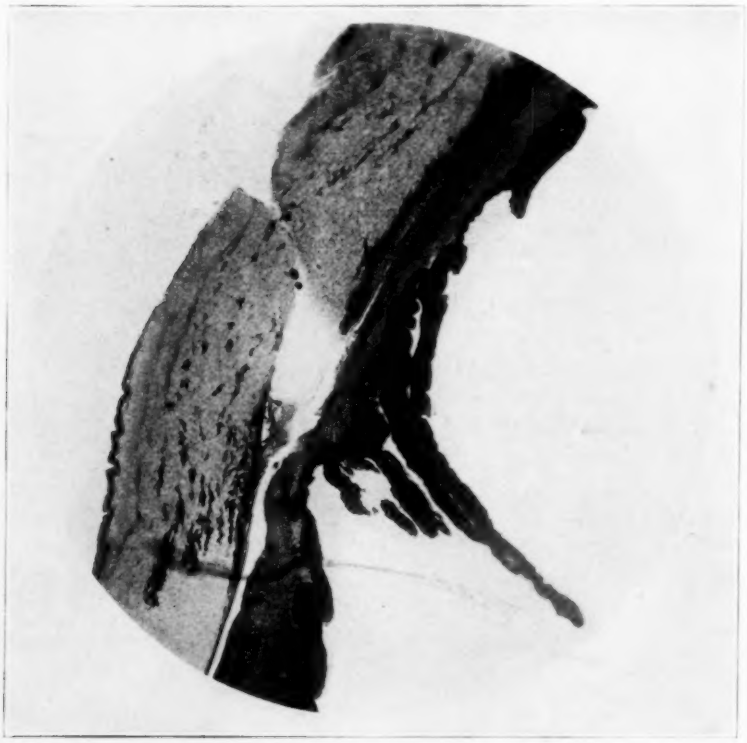

Fig. 6. Kitten's eye showing incision far back.

which lend themselves best to this method of iridectomy are acute inflammatory, the chronic simple and those forms of secondary glaucoma which resemble the chronic variety in their freedom from congestion.

Case No. 1. Mrs. B. K. Housewife. Jewess. Admitted to the Illinois Charitable Eye and Ear Infirmary, October 1st, 1923. 25 years of age.

Her vision began to fail two years previously. She had trouble in reading fine print and had frequent temporal headaches.

Family and personal history was negative except as noted. Negative Wassermann.

A case of glaucoma of the chronic simple type; cupped discs; contracted visual fields, and tension of 42 in each eye. Vision was 20/25 in each eye. Was treated with eserin for 6 weeks, but tension would rise after its withdrawal.

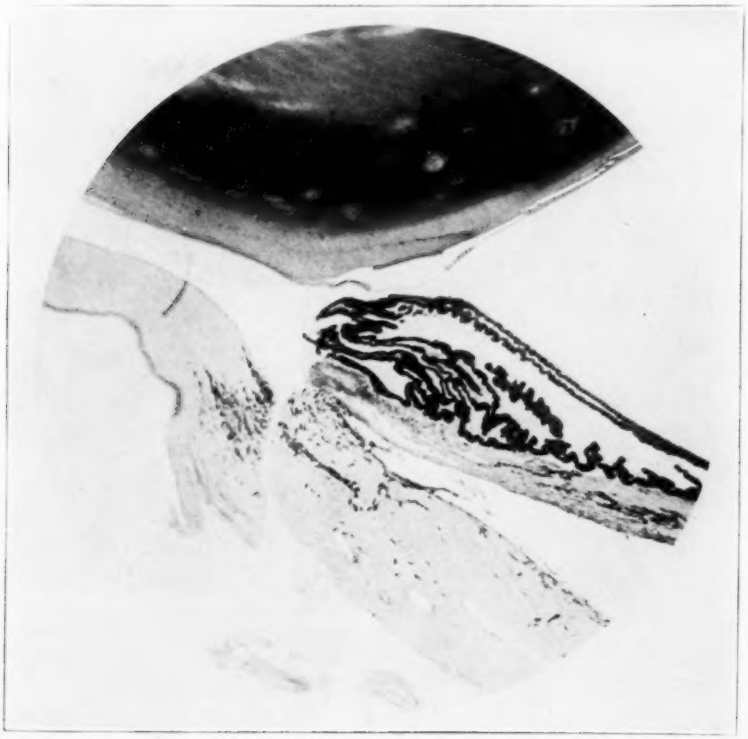

Fig. 7. Complete basal iridectomy in kitten's eye.

One month after iridectomies, the tension was 13½ in the left eye and 23 in the right eye. At the time of this writing, nearly three years after operation, the tension was 15 in the left eye and 18 in the right eye. Her vision was 20/20 with correction in each eye. Her visual fields were enlarged. In neither eye was there any sign of a filtering cicatrix.

It is interesting to note, also, that the eye with the wider coloboma, the right eye, had persistently the higher tension. The tension was taken many times in the three years, and while there was always some flunctuation, it was always higher in the eye with the wider coloboma. In the right eye the visual field enlarged from 20 to 60 in the upper temporal quadrant. In the left eye it enlarged in the upper quadrant from 35 to 60. The lower fields were limited to 15 in both eyes, and were not enlarged following the operations.

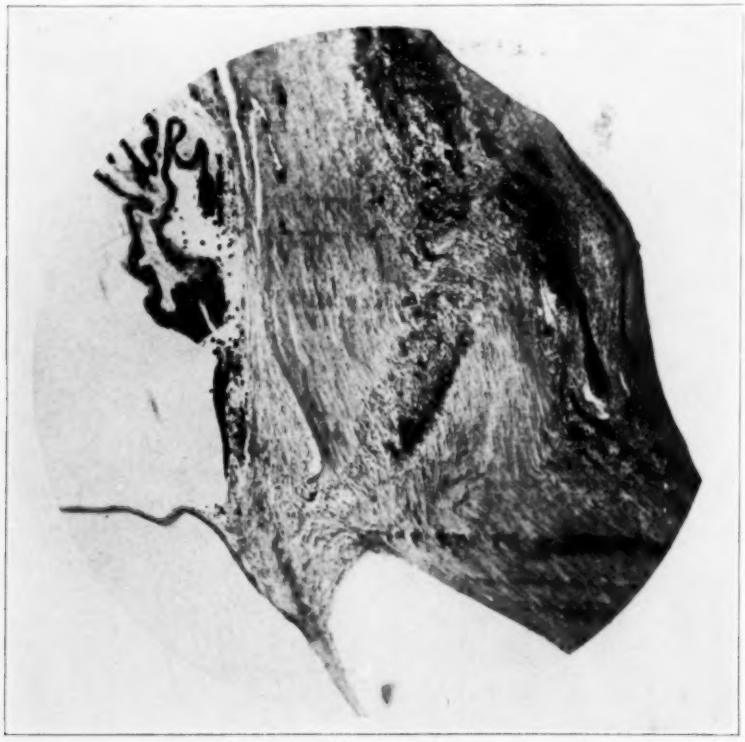

Fig. 8. Slide from laboratory of the Illinois Eye and Ear Infirmary, showing inner margin of incision far forward from the canal of Schlemm. Iris tissue left between wound and apex of the angle.

Case 7. M. H. Patient of Dr. Lloyd of Hammond, Indiana. Age 26. His left eye was injured with foreign body six months before, which resulted in a perforating ulcer. This was later followed by glaucoma, with vision reduced to fingers at 18 inches. Temporary relief was obtained with eserin. Iridectomy was performed with a keratome incision, January 16, 1924. After the tenth day, the tension was 45 mm. January 26, 1924, a deep iridectomy reduced the tension to 20, with vision of counting fingers at 3 feet, which condition is present at this writing.

Case 8. Helen H. Patient of Dr. Boettcher of Chicago. Age 15. Secondary glaucoma following chronic inflammation of ciliary body and choroid. No light reflex and no perception of light.

It was suggested, that before enucleation was done, an attempt be made to reduce tension by deep iridectomy. This

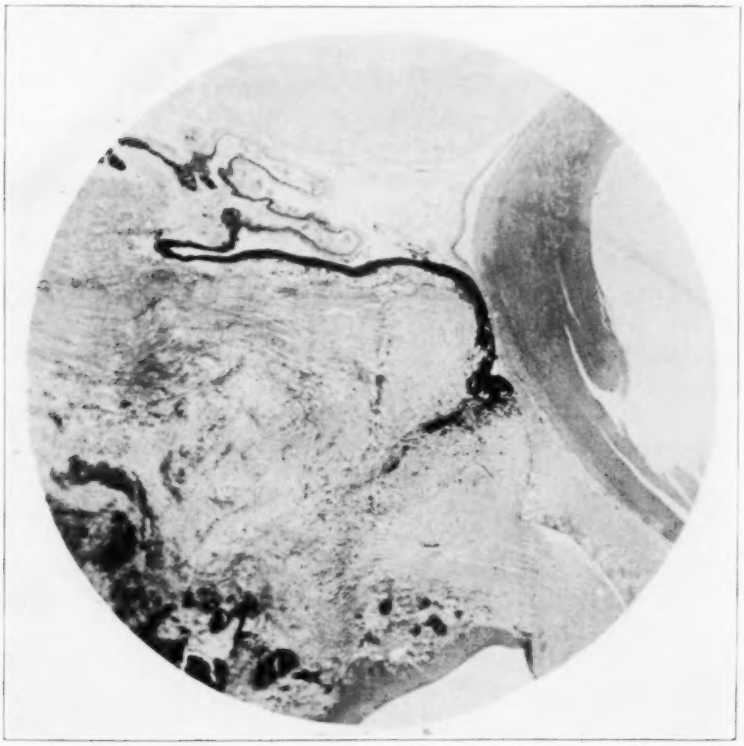

Fig. 9. Case No. 30. Glaucoma following thrombosis of central vein. Incision could not be made farther back.

was done January 28, 1924, with a very temporary result. The eye was enucleated and found to contain extensive ossification of the choroid.

Case 25. L. L. An Infirmary patient. Age 49 yrs. Chronic simple glaucoma in both eyes. Negative Wassermanns. Physical examination also negative. Right vision 16/200; left vision, light perception. Deep iridectomies followed by choroidal hemorrhage and loss of both eyes.

Case 30. S. J. J. An Infirmary patient. Age 64 yrs. Secondary glaucoma following thrombosis of central vein of the retina. Attempted deep iridectomy. Tension rose from 45 to 65, and enucleation was necessary. A microscopic slide shows that the incision was not properly made. It probably could not be made in the angle.

Fig. 10. Case No. 62. Large coloboma above from operation 23 years before. Small peripheral opening in the iris below.
Fig. 11. Case Nos. 58 and 59. Two openings in the iris. The smaller and less peripheral made with knife needle (Curran operation). Larger peripheral iridectomy necessary to reduce tension permanently.

Case 58-59. F. C. W. Age 70. Patient of Dr. Trimble of Emporia, Kan. Always near sighted. Vision failing for five years. Glaucoma diagnosed at that time, two years ago. Curran iridotomy performed in both eyes. At the time of the examination January 27, 1926, right vision finger four inches; left vision 8/200; right tension 44; left tension 34. After double peripheral iridectomies January 28, 1926, tension R. 26; L. 18. The drawing shows the appearance of the iris. There has been no further loss in vision and tension remains as noted.

Case 61. Mrs. Minnie Baker. Age 62. Patient of Dr. Dale of Chicago Heights. Secondary glaucoma of right eye following iritis 9 years previously.

An acute attack five or six weeks previous to March 7, 1926. T. 95.

Deep iridectomy under general anesthetic March 10, 1926. Relief for 12 days. Tension was then 78. August 16, 1926, eye quiet and free from pain. Staphyloma present. T. ++.

Case 62. Mrs. J. W. C. Age 47. This patient has but one eye, and it had been operated upon 23 years previously for glaucoma. A drawing shows the large coloboma above and the small peripheral opening in the iris below. She had retained good vision for the 23 years, but had recently been conscious of some failure. The tension was 64. Deep peripheral iridectomy was performed March 17, 1926, below. Tension is now 14, with definite signs of external drainage. The vision is the same, 6/10, but the field has enlarged 15° on the temporal side.

Summary of Cases.

33 Cases of chronic simple glaucoma.
(Failure in two, from choroidal hemorrhage, about 94% successful in reduction of tension to well within normal limits.)

6 Cases of acute inflammatory: 100%.

6 Secondaries in aphakia or following traumatism: 100%

8 Secondaries to iritis: Results good in 7.

14 Cases of chronic inflammatory in varying stages, or chronic simple with acute exacerbations: 4 failures.

67 Cases of different types: 7 failures, 90% successful in reduction of tension.
Definite signs of external drainage in 6 cases, about 9%.

Conclusions.

The iris can be removed at its root if the incision can be made as described.

Just as good results can be obtained in the reduction of tension by the removal of only a small section of the ciliary border.

Acute inflammatory glaucoma, chronic simple glaucoma, and some forms of secondary glaucoma which are acute or are free from congestion yield good results.

	Patient.	Sex.	1st Examination.	Age.	Eye.	Type.	Duration.	Vision.	Tension.	Operation Date.	Tension 3 Mo. After.	Late Tension.	Late Vision.
1	B. K.	F	10/ 1/23	25	L.	C. S.	2 Yrs.	20/25	42	11/16/23	13	15	20/20
2	B. K.	F	10/ 1/23	25	R.	C. S. (Sec.)	2 Yrs.	20/25	42	12/14/23	23	18	20/20
3	L. H.	M	11/15/23	83	L.	(Aph.)	6 Mo.	Shad.	54	11/22/23	N	11	Shad.
4	M. P.	F	11/21/23	28	L.	C. S.	5 Yrs.	L. P.	55	11/23/23	N	18	L. P.
5	C. H.	F	11/27/23	57	R.	A. I. Sec.	7 Da.	L. P.	70	12/ 2/23	N	19	25/65
6	M. J.	F	7/16/23	73	R.	Aph.	5 Mo.	L. P.	34	12/ 7/23	N	16	L. P.
7	M. H.	M	1/16/24	26	L.	S.	8 Mo.	F-18 in.	45	1/26/24	N	20	F-3 Ft.
8	H. H.	F	1/25/25	15	L.	S.	?	0	++	1/28/24			Enucleation
9	Mo.	M	1/ /24	30	L.	S.	?	0	80	1/ /24	N	30	0
10	A. B.	M	11/12/25	66	R.	C. S.	4 Yrs.	1/200	36	11/21/24	16	17	1/200
11	A. B.	M	11/12/24	66	L.	C. S.	4 Yrs.	9/200	25	11/30/24	15	15	9/200
12	C. G.	F	11/ 6/24	45	R.	C. S.	4 Yrs.	0	32	11/24/24	15	18	0
13	C. G.	F	11/ 6/24	45	L.	C. S.	1 Yr.	4/200	39	12/12/24	18	21	20/120
14	M. S.	F	11/12/24	62	L.	C. A.	3 Mo.	L. P.	+++	11/13/24			Enucleation
15	M. B.	F	11/26/24	61	L.	C. A.	1 Yr.	6/6—	55	11/28/24	26		
16	M. R.	F	8/12/24	54	R.	Sec.	3 Yrs.	6/30	37	8/15/24	22	19	6/10
17	M. R.	F	8/12/24	54	L.	Sec.	3 Yrs.	6/20	28	9/ 1/24	21	19	6/12
18	A. B.	M	5/19/24	39	R.	S. A.	24 Hrs.	?	+++	5/20/24	16	15	6/6—
19	M. S.	F	9/20/24	60	L.	S. A.	?	6/30	37	12/12/24	17	24	6/12
20	T. R.	M	12/ 3/24	64	L.	C. S.	1 Yr.	2/200	36	12/12/24	15	12	2/200
21	J. W.	M	11/ 3/24	66	L.	C. S.	1 Yr.	6/120	39	12/19/24	23	14	6/20
22	F. J.	M	10/ 1/24	59	R.	C. S.	4 Yrs.	L. P.	40	1/12/25	25	27	L. P.
23	W. H.	M	12/29/24	49	L.	C. S.	4 Yrs.	0	56	1/ 2/25	13	15	0
24	W. H.	M	12/29/24	49	R.	C. S.	2 Yrs.	H-3 in.	42	1/ 2/25	10	8	
25	L. L.	M	1/29/24	49	R.	C. S.	1 Yr.	16/200	51	1/12/25			Choroidal Hemorrh.
26	L. L.	M	1/ 9/24	49	L.	C. S.	2 Yrs.	L. P.	53	1/12/25			Choroidal Hemorrh.
27	J. F.	M	9/ 3/24	70	L.	C. S. A.	9 Mo.	F-3 Ft.	45	9/ 6/24	10	8	F-3 Ft.
28	C. F.	F	1/26/25	69	L.	C. S.	8 Mo.	0	60	1/30/25	21	21	0
29	G. R.	M	1/30/25	41	L.	Sec.	18 Mo.	0	42	2/ 2/25	19	19	0

						Sec.								
30	C. J.	M	2/ 9/25	64	L.	Ins.	2 Wks.	L. P.		45	2/ 9/25	65		Enucleation
31	J. S.	M	2/27/25	43	L.	Acu.	4 Da.	F-4 Ft.	+++		3/ 2/25	23	23	6/7
32	J. S.	M	2/27/25	43	R.	Acu.	1 Da.	6/7	+++		3/ 7/25	23	26	6/7
33	S. S.	M	2/13/25	68	L.	C. S.	35 Yrs.	F-6 in.		69	4/13/25	22	23	F-6 in.
34	W. S.	M	3/ 4/25	72	R.	C. S.	2 Yrs.	L. P.		80	4/13/25	17	20	L. P.
35	W. I.	M	3/19/25	46	L.	C. S.	1 Yr.	0		60	4/13/25	25	23	0
36	W. I.	M	3/19/25	46	R.	C. S.	6 Mo.	5/200		50	4/13/25	28	25	5/200
37	M. B.	F	3/16/25	52	L.	C. S. A.	8 Yrs.	6/15		65	4/17/25	20	20	6/7
38	M. M.	F	5/ 5/25	67	L.	Sec.	3 Wks.	L. P.	++		5/ 6/25	25	24	L. P.
39	J. C.	M	4/15/25	54	R.	C. S.	1 Yr.	20/200		53	5/ 6/25	20	17	6/15
40	J. C.	M	4/15/25	54	L.	C. S.	?	6/6	+++		6/ 3/25	10	12	6/6—
41	M. C.	F	6/11/25	78	R.	C. S. A.	1 Yr.	0	+++		6/12/25			Enucleate. Ch. H.
42	M. C.	F	6/11/25	78	L.	C. S.	?	10/70	+		6/12/25	10	21	6/12
43	R. H.	M	9/ 5/25	40	L.	A. S.	2 Da.	?		33	9/10/25	30	25	F-3 Ft.
44	M. K.	F	10/23/25	35	L.	A. S.	6 Yrs.	0	+++		10/23/25	N		
45	M. K.	F	10/23/25	35	R.	Acu.	5 Da.	6/6	+		10/23/25	N		
46	A. G.	F	10/12/25	42	L.	C. S.	5 Mo.	H. M.		37	10/19/25	22	22	Hand Mov.
47	G. M.	F	10/13/25	49	L.	C. S.	?	0		57	10/30/25	18	10	
48	J. S.	M	10/23/25	72	L.	C. S.	Mons.	P. & P.		39	10/23/25	14	23	L. P.
49	J. E.	M	10/20/25	53	L.	A. I.	6 Da.	5/200		54	10/30/25	17	18	5/200
50	P. H.	M	9/30/25	38	R.	C. S.	1 Yr.	0		51	11/ 6/25	24	25	0
51	M. M.	F	10/26/25	74	L.	C. S.	3 Yrs.	0		38	11/ 6/25	10		
52	R. C.	M	11/24/25	29	L.	S. A.	1 Yr.	0		65	11/25/25	31	25	
53	A. B.	M	12/ 7/25	65	R.	C. I.	2 Yrs.	0		47	12/14/25	25	25	0
54	A. B.	M	12/ 7/25	65	L.	C. S.	5 Wks.	20/50	+++		12/16/25	10	8	
55	M. B.	F	1/22/26	67	R.	C. S.				53	1/22/26	16	16	
56	M. B.	F	1/22/26	67	L.	C. S.				65	1/22/26	18	18	
57	M. A.	F	1/18/26	79	R.	C. S.		F-3 Ft.		40	1/23/26	25	25	F-3 Ft.
58	F. N.	M	1/27/26	70	R.	C. S.	5 Yrs.	F-4 in.		44	1/27/26	22	26	F-4 in.
59	F. N.	M	1/27/26	70	L.	C. S.	5 Yrs.	8/200		34	1/27/26	10	18	8/200
60	B. L.	M	2/ 1/26	77	R.	C. I.	2 Mo.	0		54	2/ 5/26	51		0
61	M. B.	F	3/ 7/26	62	R.	S. A.	4 Yrs.	0		95	3/10/26	65	?	
62	M. C.	F	3/17/26	47	L.	C. S.	23 Yrs.	20/50		64	3/17/26	15	14	6/7
63	J. J.	M	3/19/26	70	L.	Ch. I.	4 Yrs.	0		72	3/19/26	46		L. P.
64	M. C.	F	6/24/26	48	L.	Sec.	1 Wk.	6/60		38	6/26/26	30	27	6/15
65	A. B.	M	4/ 8/26	41	L.	S. A.	12 Hrs.	H.	+++		4/ 8/26	20	20	6/10
66	E. B.	F	3/17/26	70	L.	S.	3 Mo.	0		66	4/ 2/26	15	15	0
67	S. P.	M	7/21/26	63	L.	Acu.	5 Da.	Hand		30	7/21/26	15	25	6/30

STEREOPTICON SLIDES.

1. Table of cases.
2. Table of cases. (3 slides.)
3. Suker's drawing showing line of incision with cataract knife.
4. Reese's drawing showing line of incision with keratome.
5. Drawing modified from Salzman and Brown, showing lines of puncture and counterpuncture.
6. Showing near peripheral incision in pig's eye. Some iris prolapsed.
7. Incision in pig's eye not in extreme periphery.
8. Kitten's eye, incision far back.
9. Kitten's eye showing incision and complete root iridectomy.
10. From Parson's pathology of the eye. Glaucoma iridectomy. Incision too far forward.
11. Glaucoma case from the laboratory of the Illinois Charitable Eye and Ear Infirmary. Incomplete iridectomy.
12. Attempted deep iridectomy in secondary glaucoma following thrombosis of central vein. Case 30.
13. Only peripheral portion of iris removed. Case 58 and 59. A previous iridotomy had been performed. (Curran method.)
14. Only small portion of periphery of iris removed below on account of previous large coloboma above. Case 62.
15. Summary of cases.

Microscopic slides prepared in the laboratory of the Illinois Eye and Ear Infirmary by Dr. Theobald, assistant pathologist.

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DISCUSSION.

DR. JAMES W. MAY, Kansas City, Mo.: The brilliant results obtained by Dr. Woodruff with the operation of deep iridectomy (90 per cent successful in the reduction of tension), would lead one to believe that glaucoma is about to be relieved of its terrors. However, other operations such as iridectomy, the various anterior sclerectomies (as Lagrange and Elliot), altho still maintaining prominent places in the operative treatment of glaucoma, have not become panaceas. Therefore, until it has been more universally used and has stood the test of time, it cannot be given a permanent place in the treatment of glaucoma.

My experience has been limited to the observation and care of six cases at our clinic in Kansas City operated by Dr. Woodruff, and in these the results in his summary are borne out. The vision has remained the same or slightly better in all cases, and the tension well within the normal range, 22 to 32 McLain. The last case operated, January, 1926, both eyes chronic simple, showed actual increase in vision, with tension L. E. 20, R. E. 26. Both eyes showed well defined blebs of external drainage, one being as large as seen in Elliot's trephine. If one had not known absolutely the kind of operation performed, it would naturally be mistaken for an Elliot operation.

As to features of his operative technic, the simple method of anesthesia is to be commended. The final instillation of a few drops of 10 per cent cocain, holding the lips of the wound apart and placing it upon the iris, insures the best of anesthesia. I use this method in iridectomy for cataract, and there is never any evidence of pain from the patient.

Again, if the same results can be obtained by removing such a small piece of iris in the periphery, then a marked advantage is evident, because of the maintenance of the sphincter.

In the cases under my continual observation, there was apparent fistulization in four eyes, three of which were blebs as large as one finds in the ordinary Elliot trephine. Possibly, in the other cases, a smaller, almost invisible bleb might be demonstrated. Woodruff reports 9 per cent of his cases showing signs of external drainage. This raises the question, does the operation depend upon a permanent fistulization for its success? If so, then as an active competitor the Elliot trephining must be compared.

The idea that iridectomies in many cases are performed poorly, with none of the iris root removed, can readily be proven. However, who has not seen a poorly performed iridectomy deliver brilliant results, particularly in acute inflammatory glaucoma. Lagrange contends that the beneficial results obtained from iridectomy depends upon the inclusion of iris in the incision, producing a fistulization.

The writer would like to ask just how much emphasis Dr. Woodruff places upon the use of the spatula to separate the iris at its attachment. He certainly has not dwelt to any great length on what transpires following this procedure.

Why is Török's operation better than Heinie's cyclodialysis, or why is it better than a simple iridectomy?

Regardless of whether or not one does the Török operation, the argu-

ment for the use of a cataract knife to make the incision is perfectly logical, and in all probability it will become the normal procedure.

Dr. W. H. Luedde, St. Louis: I have not had the good fortune of Dr. May to see the operation of Dr. Woodruff. I heartily agree with some of the principal points that Dr. Woodruff has made. I am glad to know of the great success of the operation in his hands. I believe, in the first place, it is well to get your incision back into the angle as far as possible, and that the root of the iris should be removed if possible. I do not believe it is as essential to remove the root of the iris as it is to get far back into the angle with your incision. In that way, an opening is secured into the space which leads to the veins in the canal of Schlemm.

I also agree with Dr. Woodruff in emphasizing the possibility—in fact, the great probability—that removal of the ciliary border of the iris will result in as much effect in the reduction of tension as will a large iridectomy, and, as Dr. May emphasized, will retain the activity of the sphincter muscle.

As to the matter of making the incision, I beg to suggest a different point of view. I presented some four years ago, in Washington, a compilation of various things I had learned about getting into the eye in glaucoma, and well back into the angle. First raise a large conjunctival flap as proposed in Elliot's later method for trephining. You may, if you will, split the cornea at the limbus. Then, make a tiny nick with a keratome into the anterior chamber. Thus you will get a little valve like opening that will permit you to reduce gradually the tension of the glaucomatous eye. When you remove the keratome, you lose perhaps a drop of aqueous; a touch on the lip of the wound will let out another drop and finally the eye has lost enough, so that when you touch it with the spatula, you realize the hypertension has been materially relieved. By relieving the tension gradually, you will escape some of the dangers of deep choroidal hemorrhage that you must have with a wide open incision. That partial initial relief has an advantage also over the Elliot trephine in opening into the anterior chamber. It is done under the conjunctival flap and the edge of the cornea may be split as in Elliot's operation, but as Dr. Woodruff has emphasized, in some of these pathologic cases you cannot locate the exact site because the angle is in a measure obliterated. After the tension is gradually reduced, I cut with a scissors each angle of the original tiny keratome incision outward and backward sufficiently to bring the incision back into the angle. In that way you have both edges of the wound out well into the angle. Then I pick up the iris and make the iridectomy.

I wish to confirm what Dr. May has said about the importance of the filtering scar. We have them after the method of incision just described. It is not necessary to make a round trephine opening in order to get a good permanent filtering scar. Dr. Patterson of this city told me yesterday he has four or five cases in which this "winged" keratome incision was used, and he will be pleased to show them to anyone interested. I do not believe it is the intention of Dr. Woodruff to do away with the advantage of the filtering scar.

We have three ways in which drainage from the anterior chamber in glaucoma can be achieved. One is thru the iris backward; another is into the suprachoroidal space, and the other is subconjunctivally.

Mr. Treacher Collins, in speaking of the operation I have just described, said the success of any such operation depended upon the thoro division of Descemet's membrane. He believed the Elliot operation opened the membrane more thoroly than any other. If you get the tip of the scissors into the anterior chamber and cut thru, you have every reason to suppose that you have divided Descemet's membrane very thoroly.

The next point Mr. Collins established is, that the absence of infection is essential to the success of any operation for glaucoma. For that reason I insisted on operating under a large conjunctival flap, as Colonel Elliot suggested in later years. Mr. Collins' explanation was, that when infection is present, the edges of the wound, both in the cornea and sclera, become covered with cicatrix which prevent drainage. If no infection was present, these tissues did not heal with a firm cicatrix and a certain amount of filtration permitted ultimate success.

I am sorry that I cannot qualify by actual experience and personal contact with the cases operated as Dr. Woodruff has described, but his success is a better evidence of its value than any words of mine can add.

Dr. Harold Gifford, Omaha: The wonderful success the doctor has had in reducing the tension in these cases is the most striking thing about the operation. One of the important factors in explaining Dr. Woodruffs' results, is the fact that the iridectomy is done subconjunctivally. Great stress is laid on that by several men. I have noticed one or two articles recently which say that that is the whole trick of the Lagrange sclerectomy, the large flap. The scleral incision does not heal with ingrowth of epithelium, if the thing is left in such shape that drainage takes place beneath this flap. It occurred to me many years ago that we ought to prevent this growing in of the epithelium. Following out this idea, I made the puncture well out from the limbus, passed the knife subconjunctivally thru the limbus, then out beneath the conjunctiva of the opposite side, allowing the aqueous to escape beneath the conjunctiva. This made such a bleb that it was easy to finish the scleral flap without penetrating the conjunctiva again; so that the latter was injured only at the site of the first puncture. I thought that was ideal, but I did not get anything very remarkable in the way of results. The tension came back. Angelucci advocated practically the same operation for the purpose of freeing the angle. His idea was to cut thru the adhesions between the iris root and the cornea, but I never heard that his results were anything great. But the combination of the subconjunctival incision with the incision of the iris clear up to the root may be the secret of Dr. Woodruff's success, for an opening thru the iris at the extreme periphery of the chamber certainly has more effect on the tension than if the opening is a millimeter or so nearer the center.

Dr. H. W. Woodruff, Joliet, Ill., (closing): I did not know that Dr. May's cases, which he was kind enough to allow me to operate, showed such a high percentage of external drainage as he reports. This will make it necessary to modify my percentage of cases showing external drainage.

The case which I have just told Dr. Gifford about has been under critical, frequent examinations during nearly the last three years. The consensus of opinion has been that it showed no sign of external drainage.

Even tho we should raise the number to 25 per cent, a statement made by some observers, still there is left 75 per cent in which the drainage must be by the internal route.

Dr. May asks about the use of the spatula. In the early cases of glaucoma it is not necessary, but is of use in those cases in which the incision cannot be made far back on accont of contact of the iris with the cornea.

I approve of Dr. Luedde's idea of gradual reduction of tension if it can be brought about. It is quite evident that it would lessen the danger of choroidal hemorrhage, but the idea of extending the incision and making the angular cut backward, I am not so keen about, because it necessitates the introduction of some instrument into the eye, and I think that is an objection also to Dr. Vail's modification of the Elliot trephine. I have found it an objection in attempting to enlarge the opening with some cutting instrument, a punch, for instance, that the inner blade of the punch going into the interior of the eye is a frequent cause of cataract. That is the great advantage of the incision with the cataract knife, where you place the knife well up in angle and the incision is started before the aqueous escapes.

I do feel very strongly that the leaving of any part of the iris is very likely to defeat the end of the operation. Dr. Gifford spoke of possibly one millimeter of iris being left without harm. When you consider the measurements you are dealing with here, and the very small width of the angle, that in its widest part it is less than one quarter of a millimeter, you can see that the least bit of iris, unless it is retained in its proper position in the anterior chamber, would block the angle again. The microscopic sections which I have demonstrated show that in these the incision was too far forward. I would like to know if any one has ever seen in the laboratory a microscopic section in which the iris was removed at its base. I have never seen one myself.

THE OCULOCARDIAC REFLEX.

First Communication.*

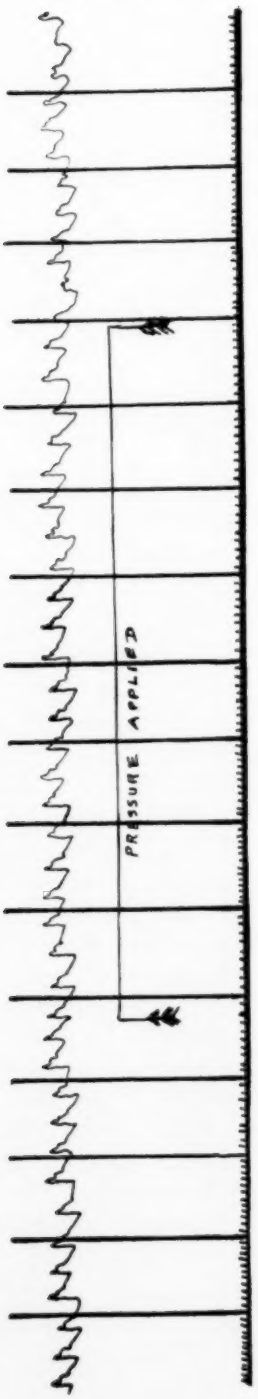
HARRY S. GRADLE, M.D.

CHICAGO.

When pressure is made upon the eyeball, there results a definite slowing of the heart rate. This phenomenon, known as the Oculocardiac Reflex, was first described by Ashner in 1908, who investigated it from both the clinical and experimental standpoint. But his research lacked the fine technic of modern methods, and he was able to draw only gross conclusions. Subsequent to this first communication, but little concerning the oculocardiac reflex appeared in the literature for some years. Then the cardiologists began to write about the clinical application of the reflex and numerous references appeared, especially in the French literature. But none of these dealt with the experimental determination of the limits and course of the reflex. Many of these references are included in the appended bibliography, altho the material contained therein does not appear in this paper.

Castelnau used the oculocompressor of Roubinovitch, with which a pressure of 18 cm. was applied to the eye for 30 seconds. The radial pulse was counted for 5 seconds preceding the pressure, again while the pressure was applied, immediately subsequent to the release of the pressure, and until the normal pulse rhythm returned. It was found that 18 cm. was the most agreeable pressure applied, altho the reflex appeared equally well with pressure of from 10 to 25 cm. The rate of the heart was slowed from two to seven beats per minute. Galup utilized not only the manometric oculocompressor of Roubinovitch, but also the oculocompressor à ressorts (spring pressure) of Bain. Pressures of from 600 to 800 grams yielded

*From the Heart Station of Michael Reese Hospital, Chicago. I wish to express my appreciation of the whole hearted assistance given in this work by Miss Howard, who is in charge of the Heart Station.


PRESSURE APPLIED

the best results. In one case, a pressure of 400 grams slowed the pulse 13½ beats a minute, and a pressure of 700 grams for 10 minutes slowed the pulse 33 beats per minute. Both the method of applying pressure as well as counting the heart rate were of the gross type, and no scientific accuracy can be attached to these results. Petzetakis measured the pulse rate with the polygraph and made pressure upon the eyeball with his fingers, both hopelessly inaccurate. Meyer measured the pulse rate with the electrocardiograph, but made the necessary pressure upon the eyeball with his fingers, thus vitiating any scientific delimitation of the oculocardiac reflex. Fribourg-Blone and Hyvert made an extensive series of measurements of the oculocardiac reflex from the clinical standpoint, but there is no clear record of the method of producing pressure upon the eyeball, or the amount of pressure employed, nor are any norms given.

In view of the lack of accurate measurements of the lower limits of the physiologic oculocardiac reflex, it was determined that the norms must be established before the path of the reflex could be studied. Practically all of the work to date has been done by cardiologists, or by physiologists, investigating from the standpoint of the heart action. The origin of the reflex, the eye, has suffered the fate of a stepchild and been neglected. A very small amount of experimental work was sufficient to show that the easily applied polygraph was too crude to yield accurate information, as can be seen from the attached tracing. Fig. 1. So recourse was had to the electrocardiograph to obtain an accurate uninterrupted record of the heart rate. One electrode was attached to the left forearm and the other to the right, and the resultant current variabilities stepped up by the ordinary clinical electrocardiograph. An arclight was used and the shadow of the string cast upon continuously advancing bromid paper. Time was recorded upon the paper in twenty-fifths of a second by the shadow of a revolving wheel, actuated electromagnetically from a tuning fork. The incidence and release of pressure upon the eyeball was recorded upon the paper by the shadow of an indicator, actuated electromagnetically from a switch in the hands of the operator.

The question of applying graduated pressure upon a given area on the eyeball over a definite length of time was somewhat more complicated. After various experimental devices

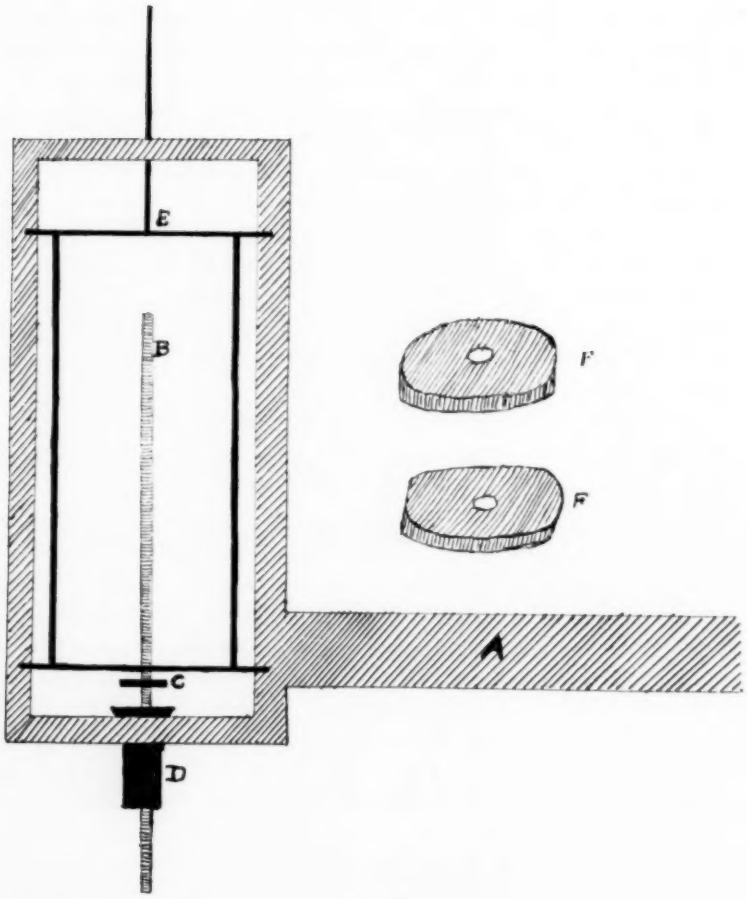


Fig. 2.

A—Rigid wooden frame.
B—Moveable stylet. 2mm. diameter.
C—Fixed stop on stylet.
D—Housing thru which stylet moves and is held vertically.
E—Sliding guide frame to lift weights on and off stylet.
F—Perforated 10 gram weights.

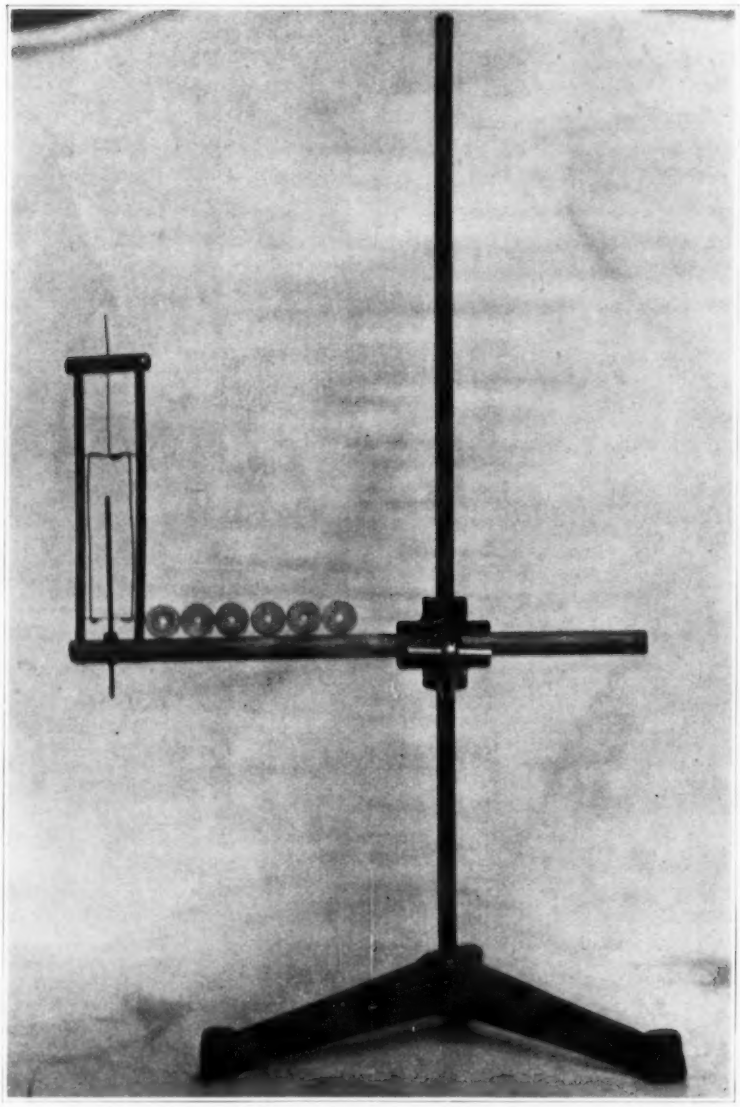


Fig. 2A.

had been tried, the following scheme proved satisfactory (Fig. 2, 2A, 3, and 4). The rigid wooden frame A was held vertically over the eye by means of a burette stand and clamp. In the base of the frame is a metal housing D, thru which glides the vertical stylet, B. The diameter of the latter is 2 millimeters. Above the housing is a metal stop, C, fixed on the stylet. The light metal frame, E, glides vertically on the rigid wooden frame, and also moves independently of the stylet. The metal weights, F, are 10 grams each and fit on the stylet, B. The weights can be lifted by means of the frame, E, so that they do not rest upon the stylet. But when the frame, E, is lowered, the weights rest directly upon the stop, C, and hence are applied directly thru the stylet to the eyeball. The photographs, Figs. 3, 4, show the device applied

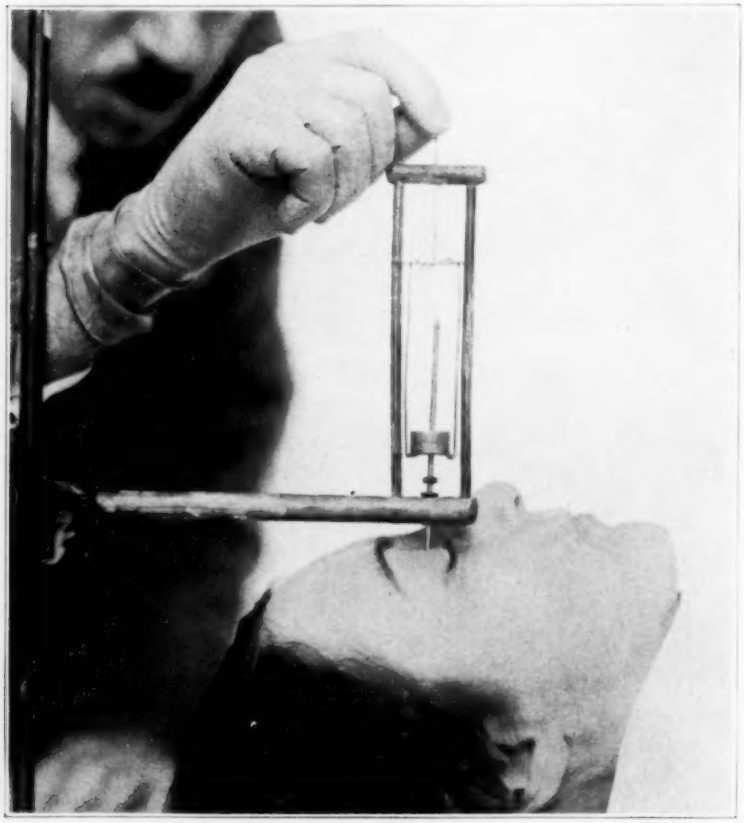


Fig. 3.

to the eyeball thru the closed lids. In the first position, the frame, E, is lifted by the hand of the operator, so that the only weight upon the eyeball is the negligible weight of stylet. In the second position, the frame has been lowered, so that the weights rest upon the stop and are transmitted directly to the eyeball. In actual practice, the operator's hand must be encased in a rubber glove, otherwise the currents generated by the muscle action of the hand and arm of the operator are recorded by the electrocardiograph.

The technic used in the various experiments varied somewhat according to the character of the experiment, but in general followed the same basic principles. The electrodes of the electrocardiograph were fastened to the right and left forearm of the subject by wet gauze bandages, and a reading of the

Fig. 4.

normal heart rate taken with the subject in a recumbent position. Then the pressure device was adjusted over the eye and another record of the heart rate made, for about ten seconds. During this time, the weight lifting frame was held aloft by the rubber gloved hand of the operator, who had the signal switch in his other hand. Then the frame was lowered gently, so that the pressure of the weights was transmitted directly to the eyeball, and at the same time, the signal switch was actuated, so that the indicator signaled on the cardiogram the exact moment that weight was applied. The weights were allowed to remain the predetermined time (a stop watch was in front of the operator) and then the frame was lifted, taking the pressure of the weights off the stylet. At the same time, the signal switch was released, indicating on the cardiogram the exact moment of release of pressure. The electrocardiograph was kept in operation fifteen or twenty seconds longer in order to record the rate of recovery. During this time, there was no motion on the part of the operator or the subject. It goes without saying that the records were made in a quiet room, from which all extraneous noises or other factors that might influence the heart rate were excluded. Several minutes were always allowed to elapse before another record was made upon the same subject. Practically all of the subjects were nurses in Michael Reese Hospital, and consequently were not unduly excited by the somewhat fantastic surroundings of an electrocardiographic laboratory.

The electrocardiograms were read with the aid of a simple magnifying lens and a protractor. The length of time that elapsed between the apex of each systole was determined and then translated into rate per second. All cardiograms were plotted on crosssection paper, the abscissae representing the rate per second and the ordinates the beats. No attention was paid to the other characteristics of the cardiograms, for it is only the rate that is of interest in this connection. It is of course futile, as well as impossible, to present all of the cardiograms and the derived charts; consequently, only illustrative ones will be shown and results of the various studies made discussed.

Illustration 5 is the chart derived from the cardiograms resulting from graduated pressure on the right eyeball of one subject. The subject was flat on her back; the right eye was closed, but the left eye open, and the visual axis of both eyes

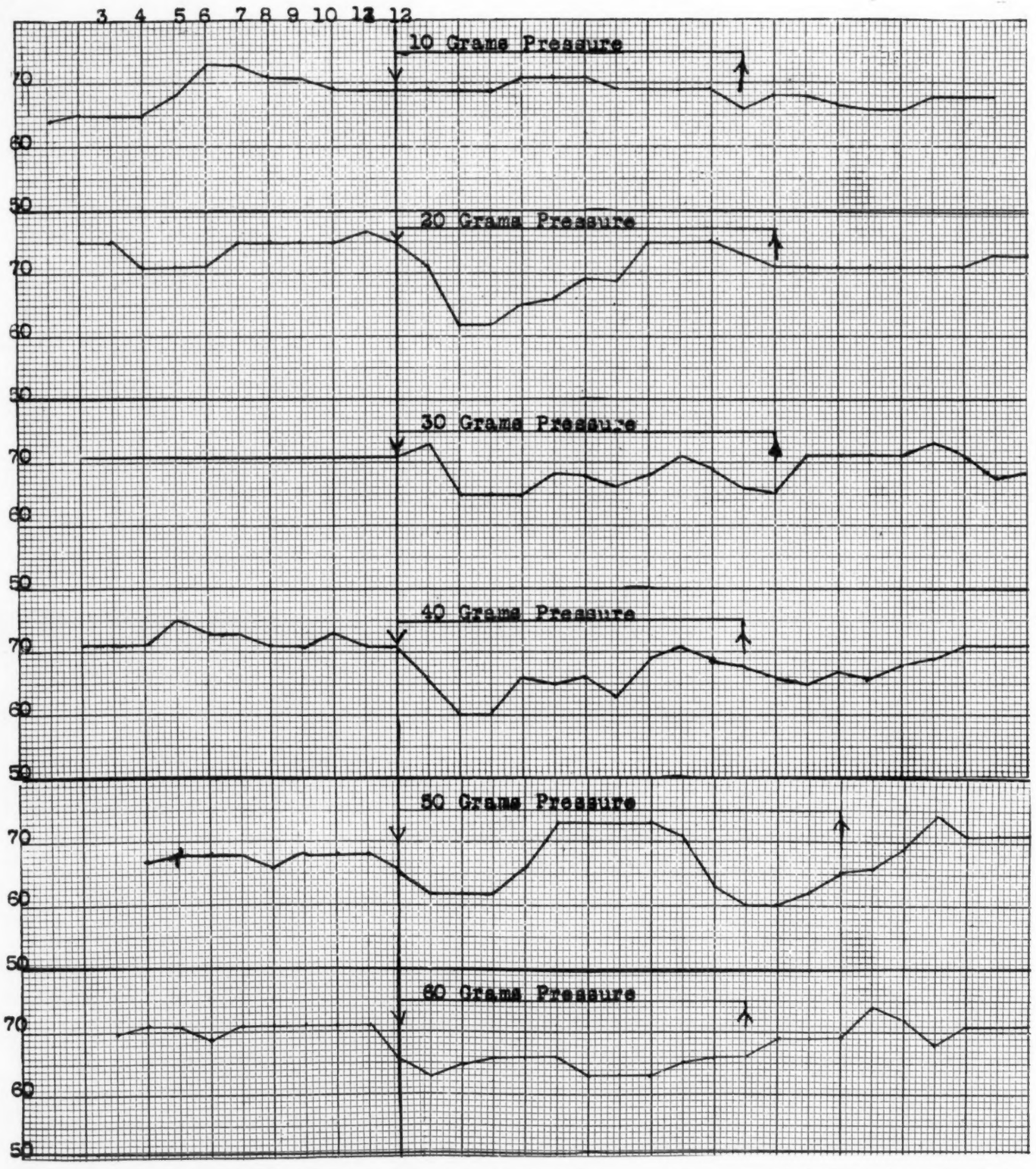


Fig. 5. Experimental subject, Miss C. Flat on back with right eye closed and left eye open. Visual axis vertical. Graduated pressure applied to center of right cornea thru closed upper lid.

was directed vertically. The stylet was applied to the center of the right upper eyelid, approximating the center of the cornea as nearly as possible. After the normal reading, 10 grams pressure was applied to the stylet and the pressure exerted upon the eye for about 10 seconds. After several minutes this was repeated with 20, 30, 40, 50, and 60 grams. There was no response to 10 grams pressure. But with all of the other weights, there was a very definite slowing of the heart rate, starting almost simultaneously with the application of the weight. Therefore it may be stated that the *minimum pressure exerted upon the eye that will produce a bradycardia is between 10 and 20 grams.* Owing to variations in the individual, as well as to variations in the oculocardiac reflex in the same individual, it does not seem possible to refine these figures. Up to 60 grams pressure, *there is no variation in the amount of bradycardia dependent upon the amount of pressure applied. The heart rate begins to slow as soon as the pressure is applied,* within the gross limits measureable with the electrocardiograph. Undoubtedly, there must be a latent period to the reflex, exactly as there is a latent period to other reflexes that can be measured accurately. For example, the latent period of pupillary contraction has been shown to be 0.1875 seconds (Gradle and Eisendrath). But the heart rate, whereby oculocardiac reflex is measured, varies from 60 to 80 beats per minute, which means that there is one beat every ¾ to 1 second. This is too gross to allow of the finer measurements necessary to determine the latent period of any reflex. At times, there is a slight increase in the heart rate for one or even two beats immediately upon application of the pressure. This is undoubtedly due to the too sudden application of the pressure and the resultant hyperstimulation of the sympathetics. The phenomenon has been particularly noticeable in excitable subjects and in those to whom the work was new and consequently somewhat fearsome. The temporary tachycardia can be seen in the graph of 30 grams pressure. *The primary bradycardia of the oculocardiac reflex persists for only one to four beats, and is followed by a fairly sudden tachycardia* that is greater than the original heart rate. This change is not uniform, and as far as can be determined, does not depend upon any known factors. In all probability, the tachycardia is due to what Howell calls "the escape from inhibition" and "the increase in the inner stimulus." The increase in the heart rate, following the primary slowing, may be equal to or greater than the heart rate previous to the applica-

tion of pressure, and it persists for a somewhat greater time than the primary slowing. But after a few seconds, a secondary bradycardia makes its appearance and remains until the pressure is removed. Then the heart rate increases slowly to a point somewhat above the normal rate for a few beats and sinks back to normal. The 50 gram graph is as typical an illustration of the oculocardiac reflex as can be found. On analysis, it can be seen that the normal rate is about 69 beats per second. Upon application of 50 grams pressure to the eyeball, the rate sinks to 61 per second for three beats and then rises rapidly to 73. It remains at the latter figure for five beats and then sinks to 60. As the pressure is removed, it rises gradually to 74 and then sinks back to 69. *The tachycardia persists for but a few beats, and is followed by a secondary bradycardia that is greater than the primary. The secondary bradycardia persists until the pressure is removed, after which the heart rate increases to above normal for a second or two.* Illustration 6 is the electrocardiogram from which the graph of 20 grams was derived.

As stated there is a great variation in the oculocardiac reflex in various individuals when tested under identical conditions. For example, eight nurses of approximately the same age were each tried with the 20, 40, and 60 gram weights applied to the right eyeball thru the closed lid. In four, the reflex corresponded much to that depicted in illustration No. 5, while in the second four, the reflex was either missing or so variable that it did not follow the average. The first four were of the placid type, with weight varying from 167 to 200 pounds each. The second four were of the excitable type, and their weight varied from 90 to 120 pounds each. The first four fell into the class designated by Eppinger and Falta as "Vagotonic," and the second four were "Sympathecitonic." Thus, *in the lower limits of the oculocardiac reflex, individuals may be classed as vagotonic or sympathecitonic, according to whether the oculocardiac reflex is present or not.* Fernald found that the oculocardiac reflex is absent in horses, cattle, sheep, and pigs. It is present to a slight degree in rabbits, but is very positive in dogs. Petzetakis, using the technic previously described, found that the reflex was more exaggerated when pressure was made upon the right eye than upon the left. This corresponds to the anatomic fact, that in the lower vertebrates, the inhibitory fibers in the vagus nerve are found almost exclusively in the right nerve, altho it is believed that in mammals they are distributed equally between both vagi. In this series of

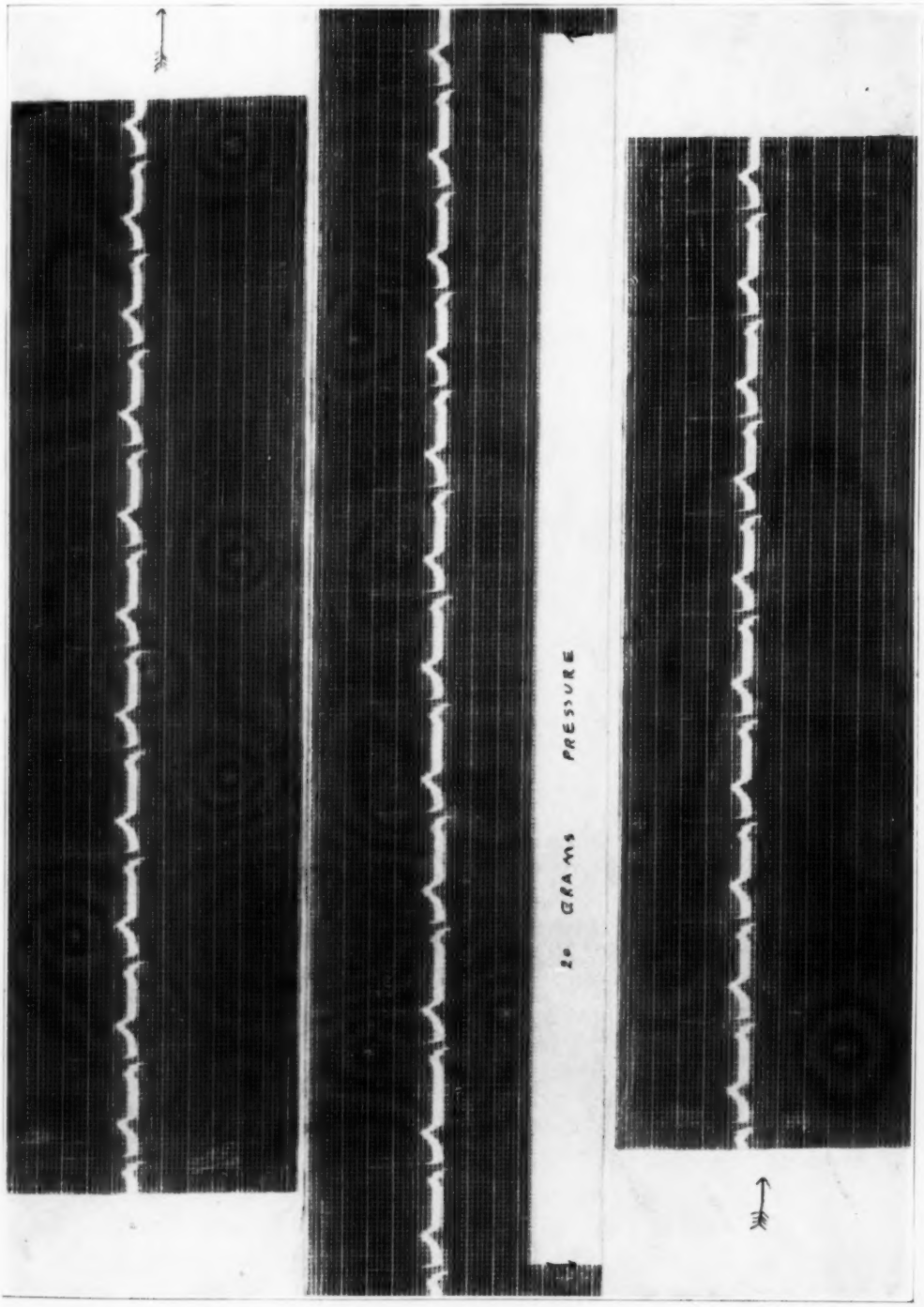


Fig. 6.

experiments, *it was not found that pressure upon the right eye produced any greater bradycardia than when pressure was made upon the left eye.* Many of the electrocardiograms of this series had to be discarded because of the irregularities in heartrate that resulted from extraneous influences, such as conversation, unexpected touching of the subject, a too sudden application of the weight upon the eyeball, unexpected sputter of the arc lamp, etc. In these cases, the regularity of the heartbeat or the variations due to ocular pressure were distorted, possibly for only one or two beats, but sometimes longer. In other words, *the stimulation of inhibition that produces the oculocardiac reflex is of a weak type, and is easily overpowered by the stimulation of acceleration that produces the common emotional tachycardia.*

The next two series of illustrations are closely correlated. No. 7 is the graph derived from electrocardiograms resulting from the application of pressure by the stylet to the orbital tissues direct without weight being applied to the eyeball. The subject lay flat upon the back with the eyelids closed and the visual axis vertical. The stylet rested upon the skin of the lower lid between the eyeball and the zygoma in such a manner, that absolutely no weight came upon the eyeball proper. *When the weight is thus upon the orbital tissue and not upon the eyeball, the minimum weight that will produce a bradycardia is 40 grams,* as contrasted with less than 20 grams when the weight is transferred thru the eyelids to the eyeball. This shows conclusively that *the exciting element of the oculocardiac reflex is pressure applied to the intraorbital contents and not to the eyelids.* Further proof is furnished by illustration No. 9. Another interesting point is that the whole process seems to be slowed down. An appreciable time elapses after pressure has been applied before bradycardia commences. This may be as much as three beats. The slowing of the heart rate proceeds at much the same pace as in the previous experiments, but the secondary tachycardia is slower in appearing and does not reach the same height. Thus, *when pressure is applied to the orbital tissue, the oculocardiac reflex is slowed in all its phases.* It would seem as tho this slowing were due to the time required for the transfer of pressure thru the tissues of the orbit. Illustration No. 8 is the cardiogram resulting from 50 grams pressure upon the tissues of the orbit as described.

Illustration No. 9 is the graph derived from electrocardiograms resulting from the application of pressure upon the eyeball direct without any intervening eyelid. The subject was lying

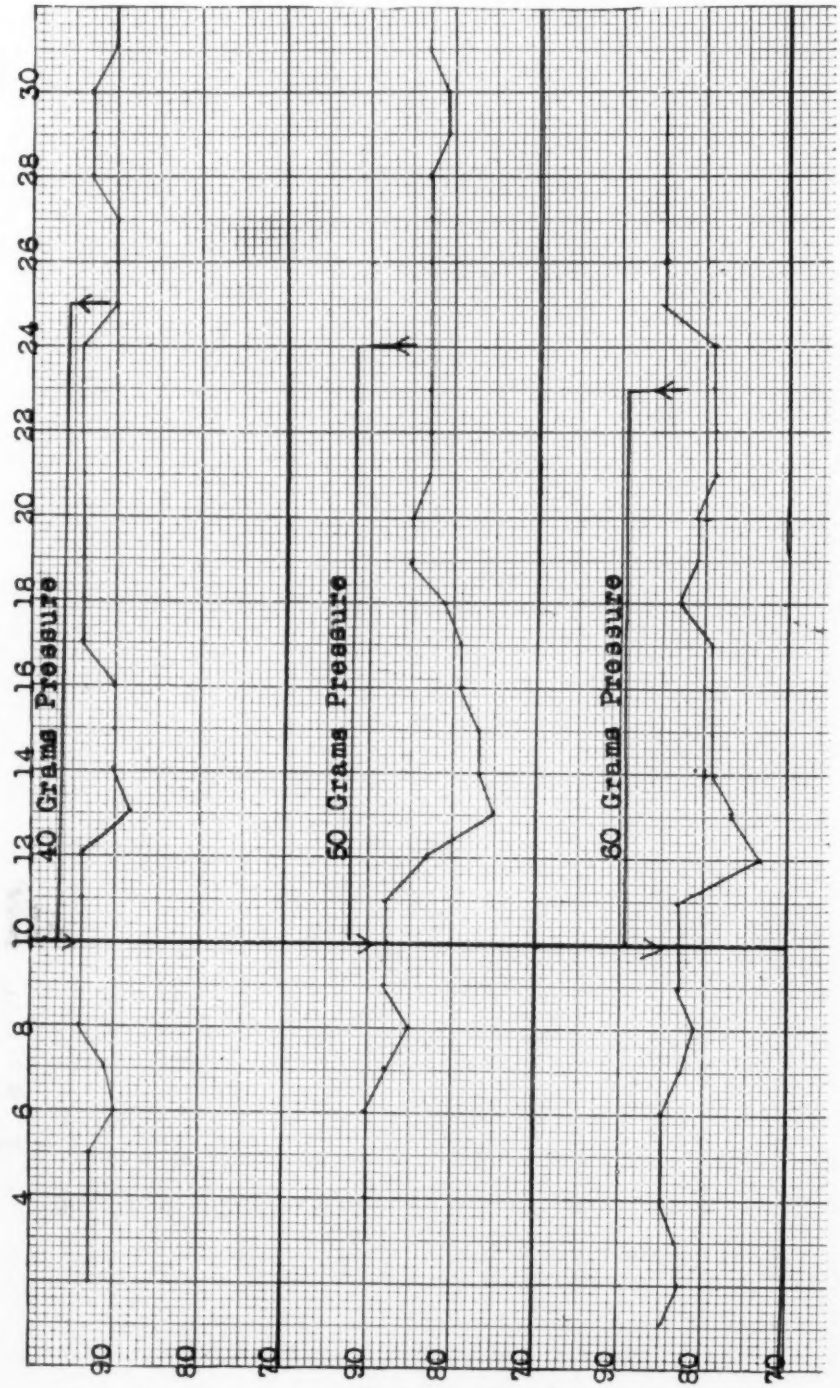


Fig. 7. Experimental subject Miss C., lying flat on back with eyelids closed. Graduated weights applied to orbit thru 2 millimeter stylet that rested upon the skin of the right lower lid between eyeball and zygoma, no weight being applied to eyeball. There was no slowing of heart rate with less than 40 grams weight.

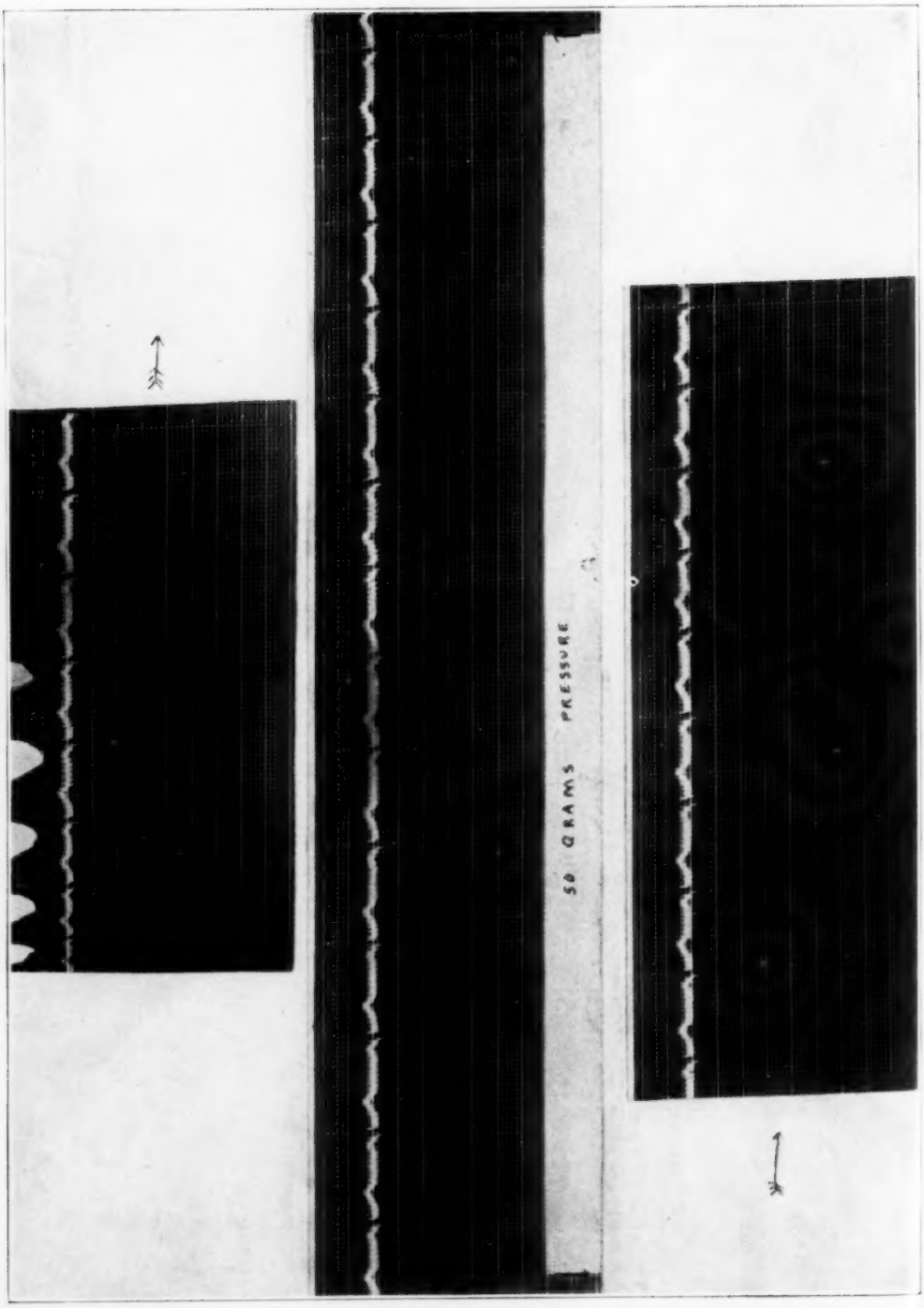


Fig. 8.

upon her left side with both eyes open and the eye rotated slightly to the left, so that the visual axes formed an acute angle with the floor. The right eye was anesthetized lightly with one drop of 2% butyn. The stylet rested directly upon the sclera, about 5 millimeters posterior to the limbus, at right angles to the visual axis, and forming an acute angle with the perpendicular. 20, 40, and 60 gram weights were applied. The resultant curves are in marked contrast to the curves of illustration No. 7. There, the reflex was sluggish: here, the reflex is sudden and sharp. It was produced by a minimum weight of 20 grams as contrasted with the minimum weight of 40 grams necessary in the preceding experiment. The resultant bradycardia is greater and there is no drag to the reflex. *Pressure applied directly to the eyeball, regardless of where, is more efficacious in producing the oculocardiac reflex than when applied thru the eyelids or to the orbit.* Illustration No. 10 is the cardiogram resulting from the application of pressure of 60 grams directly to the eyeball.

The path of the oculocardiac reflex is still a mooted question. Ever since the inhibitory action of the vagus was described, in 1845, by Edward and E. H. Weber, it has been considered that any slowing of the heart rate was due to a stimulation of the vagus. It has been shown anatomically that superior and inferior cardiac branches leave the vagus to join the sympathetics on the arch and ascending portion of the aorta to form the cardiac plexus. The vagal portion of the plexus carries the inhibitory phase, while the sympathetics that descend from the cervical region carry the accelerator phase. Thus slowing of the heart rate may be due to a stimulation of the vagus and resultant inhibition, or to inhibition of the sympathetics and diminished acceleration. The latter phase is possible in view of Howell's words, "It is important to bear in mind the general physiologic rule that afferent impulses may either excite or inhibit the activity of nerve centers." This is further borne out in the discussion of the nature of inhibition by Gaskell and Meek and Eyster. In his original article, Aschner stated specifically that the oculocardiac reflex remained after experimental section of the vagus. He also found, that with the heart exposed, excessive pressure upon the eye caused eventual stoppage of the heart in diastole, and it is a well known fact that stimulation of the cut end of the vagus can cause the heart to stop, in diastole. Thus there are two possible paths between the brain and the heart, and it is

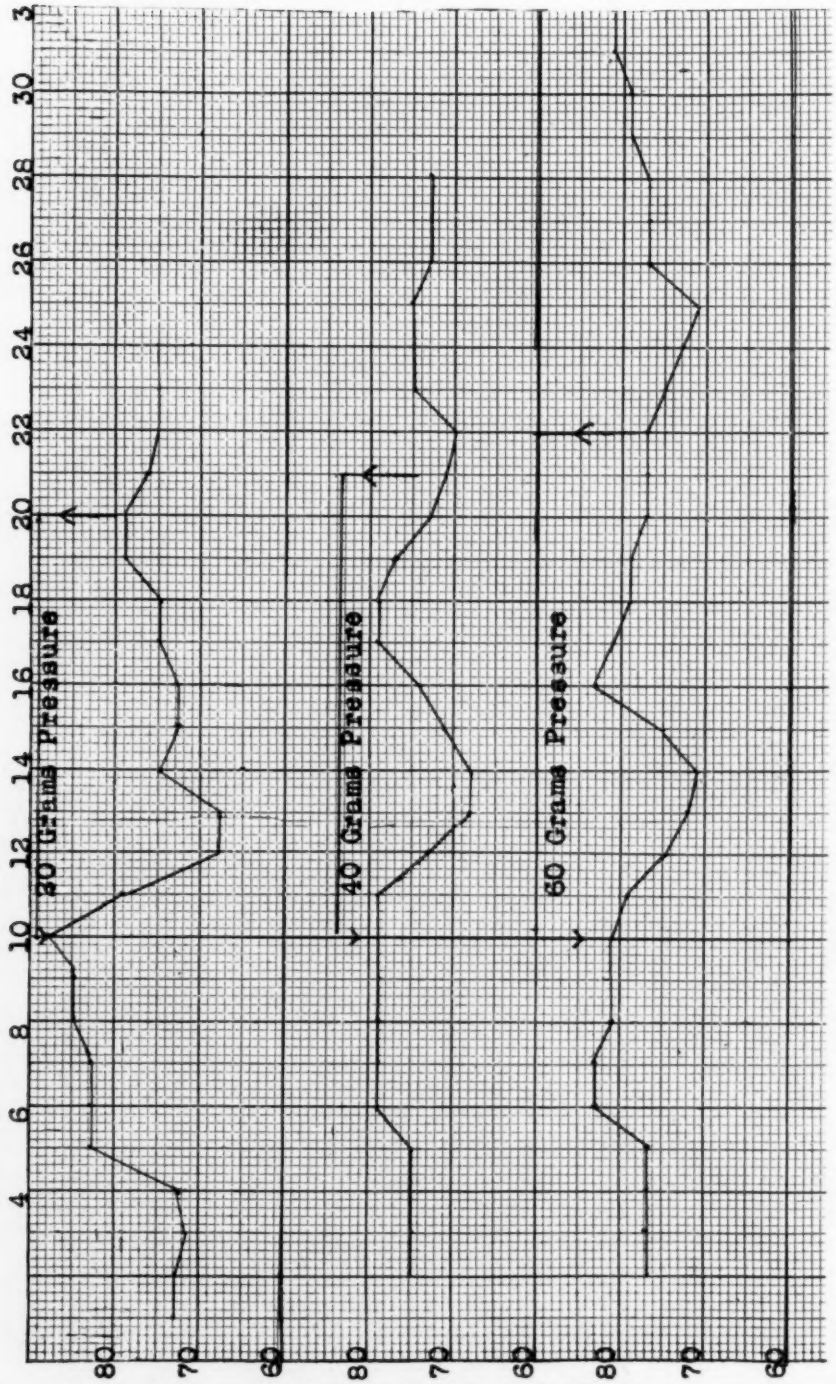


Fig. 9. Experimental subject Miss C., lying on left side with eyes open and rotated slightly toward left. Graduated pressure applied thru 2 millimeter stylet directly onto sclera, 5 millimeters posterior to the limbus and at right angles to visual axis. Eye anesthetized lightly with one drop of 2% butyn.

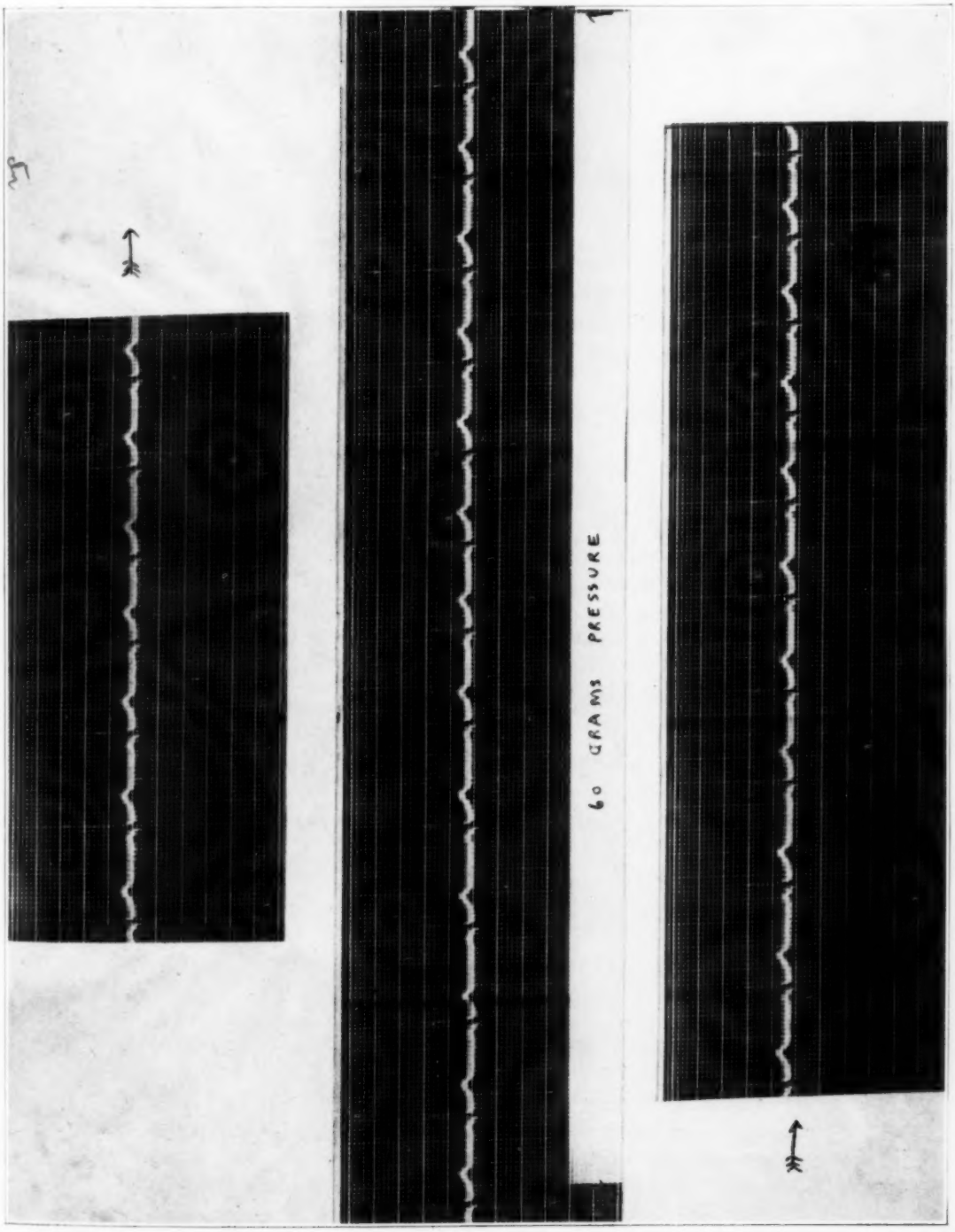


Fig. 10.

extremely probable that both share in transmitting the reflex. The inhibitory impulses that cause the bradycardia, whether they be vagal or sympathetic, undoubtedly come from the great motor nucleus known as the nucleus ambiguus, wherein lies the anatomically undelimited cardioinhibitory center. The afferent impulses to this nucleus are still undetermined. Aschner found that the oculocardiac reflex disappeared after section of one or all of the trigeminal, optic, olfactory, oculomotor, trochlear, abducens, acoustic, and facial nerves. On the other hand, Papilian and Crucearm believe that the path of the oculocardiac reflex experimentally seems to be both sympathetic and trigeminal, the latter playing the major role. Petzetakis is convinced that the trigeminal forms the afferent path. The question is open for further experimentation and ultimate clinical proof.

Summary.

From this experimental work on the lower limits of the oculocardiac reflex with an especially devised method of producing limited graduated pressure upon the eye, and recording of the resultant heart rate electrocardiographically, the following conclusions may be drawn:

1. In vagatonic individuals, pressure of from 10 to 20 grams upon the eyeball produces a slowing of the heart rate.

2. The slowing of the heart rate varies in different individuals and is not proportionate to the amount of pressure.

3. The slowing of the heart rate is followed after one to three beats by an acceleration that is equal to or greater than the normal.

4. The acceleration of the heart rate persists for three or four beats and is followed by a secondary slowing that is greater than the primary.

5. The secondary slowing of the heart rate persists until the pressure is removed.

6. After the pressure is removed, the heart rate increases to more than the normal for a few beats, and then sinks to the normal rate.

7. The slowing of the heart rate is induced more rapidly by pressure upon the eyeball direct than upon pressure thru the eyelids or thru the orbital tissues.

8. The oculocardiac reflex is induced by a weak nerve im-

pulse that is easily overpowered by nerve impulses of emotional origin.

9. These statements hold true for pressure of 60 grams or less.

10. The path of the oculocardiac reflex is still undetermined.

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DISCUSSION

DR. CLOUGH T. BURNETT, Denver: This interesting reflex has been recognized for many years. I believe it was clinically recognized long before Aschner's work in 1908. It was first employed along with other forms of vagus pressure in the attempt to relieve paroxysmal tachycardia. Commonly this consisted in pressure on either or both sides of the neck, but Continental physicians, especially, have at times employed ocular pressure, maintaining a steady and fairly firm pressure over the eyeballs, always, if effective, sufficient to be distinctly painful and usually less apt to be attended by success than vagus pressure. Morris Kahn, of New York, reported in the Journal of the American Medical Association for November 12, 1921, the production of ocular pressure by means of rubber "eye bag" which was inflated and the pressure determined with a manometer. Pressures of 100 to 320 mm. of mercury produced changes in rates in vagatonia, but in sympatheticonia there was a complete resistance to the oculocardiac reflex. Always the exact mechanism has been a matter of some conjecture, and while this excellent paper offers considerable new information and an interesting resume of the physiologic studies directed towards the determination of the path of this reflex, its exact explanation is still lacking.

In practice, it has been found that there is some difference in the type of response, according to whether the pressure is exerted on the right or the left vagus. This rests upon an anatomic basis, for the right nerve runs to the sinoauricular node, which is the pacemaker of the normal mammalian heart. The left nerve runs to the auriculoventricular node, which has to do with the conduction of the cardiac impulse from the auricles to the ventricles. The result, then, when this pure distribution obtains, is partial or complete standstill when the right nerve is compressed, and varying forms of A-V block following left vagus compression. In general, more pronounced effects are obtained when the right nerve is compressed. The inference is, that this same law obtains following ocular compression, and Petzetakis, cited by Dr. Gradle, seems to have demonstrated a greater inhibitory effect on right ocular pressure, altho Dr. Gradle does not seem to have verified this observation.

It seems to the speaker, that the changes in rate shown in these electrocardiograms are not sufficient to warrant very definite conclusions. Clinically, these changes have been most marked in conditions attended with extremely high heart rates, as paroxysmal tachycardia and paroxysmal extra systoles; here the sudden change from the high to the lower rate is marked and ofttimes abrupt.

In reference to the demonstration of a possible latent period, the employment of a high speed camera would probably be sufficient to clear up this point. As to the practical application of this work, electrocardiographic work is attended with a considerable amount of technical difficulty. The apparatus described was obviously devised for investigation purposes and is a great credit to Dr. Gradle, but when the cardiologist for any reason wishes to utilize vagus inhibition by the oculocardiac reflex path, he will be forced to do this by digital pressure and without any very definite ideas as to the exact pressure employed. Whether this can as safely be done by the cardiologist by direct pressure on the eyeball, as

suggested by Dr. Gradle, or by the older method of pressure on the lids, is a matter for the member of this branch of the profession to decide.

I wish to sound one word of caution in reference to this procedure, at least applying to the higher pressures. Sir Thomas Lewis, of London, cites one case of "serious after effects" following vagus compression. I have seen one extremely long and extremely uncomfortable standstill of the heart following right vagus pressure, altho I have never seen this occur in ocular pressure, but see no reason why it may not.

It should be borne in mind, however, that a pressure varying in different individuals, upon either the eyeball or the vagus in the neck, *may* stop the heart, usually for only short periods, but I have personally seen this to be uncomfortably long, so long, in fact, that we felt it might be permanent.

Dr. Conrad Berens, New York City: I regret that I am not able to speak from experience concerning this well devised and carefully experimental study, but there are several points which perhaps might well be emphasized.

First, the great importance of experimental investigation of mooted questions, particularly in physiology and physiopathology. It is my belief, that such studies have not been given the attention they deserve by American ophthalmologists, and that they really offer more opportunity for the advancement of the science of ophthalmology than the study of dead tissues.

The second point is the great value of graphic records which are always available for the interpretation of others, and which tend to minimize the errors of individual observation, which is a stumbling block in the scientific method. Doctor Gradle does not say that this work has a distinct clinical application at the present time, but we hope he will continue these studies, and by applying them to lesions of the nerves of the eye, important anatomic and physiologic facts may be discovered. It will be interesting to know his findings in glaucoma, and in patients with traumatic enophthalmos. I hope in the years to come the national society programs will contain more work of this same high character.

Dr. Harry S. Gradle, Chicago (closing): There is really nothing to add. This is an academic question which I undertook without any practical application in view. Eventually, something good may come out of it, but it will require a great deal more work than has been done. I have been at it for two years, and there are a great many features that are important which do not appear in this paper at all. As to the question of the right and left eye, unquestionably, when high pressure is used, the right vagus is more susceptible than the left. In the lower limits, such as were worked with in this paper, there was absolutely no difference found.

SOCIAL SERVICE AND FOLLOW UP IN OPHTHALMOLOGY. CONCLUSIONS AFTER RESTUDY OF HOSPITALS IN NEW YORK AND BROOKLYN.

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Social service department in even our most modern hospitals are of comparatively recent date, which may account for the fact that their great importance is not fully recognized by many of those who should be vitally interested.

Active interest and clear understanding of the importance of this work to ophthalmology, as shown by the literature at least, is of even more recent date. Derby,[1] in 1920, seems to have been the first to bring this to the attention of the American ophthalmologists. He reviewed the work of the Social Service Department of the Massachusetts Charitable Eye and Ear Hospital, founded in 1907, and outlined the various functions of the department in relation to ophthalmology.

In 1922, a study of follow up work was made by the Section on Ophthalmology of the Associated Out-patient Clinics of New York City thru the work of Doctor G. E. Sturges and Miss Elizabeth Tandy. This study showed the need for more follow up work in the eye clinics of New York and Brooklyn, and was the basis for recommendations made by the Section on Ophthalmology. The fact that these clinics have been recently restudied to see what effect was obtained by the recommendations made to the hospitals and clinics, is the reason why the writing of this paper was undertaken.

These studies clearly show that the need for social service and follow up work is not fully recognized, at least in New York, and we agree with Derby that "No hospital at the present day, which serves the people, can give really efficient

service unless it makes provision for social service work, and establishes some sort of follow up system."

The study of social service work and 5,200 records in five representative eye clinics in New York, at the request of the Associated Out-patient Clinics of the city of New York, in 1922, revealed the following:

Analysis of the records brought out clearly the failure of patients to attend clinic a sufficient number of times to cure the eye conditions or to prevent spread of infection. One hundred and three of the 193 cases presenting selected eye diseases are recorded as attending clinic once, 22 attended twice, 38 attended from three to five times. Of the cases with these serious diseases which might result in permanent blindness, 53.4 per cent attended the clinic only once. Only 15.5 per cent attended more than five times.

The same point is brought out still more forcibly in the instances of certain especially serious diseases. Eighty-three per cent of the cases of purulent conjunctivitis, 80 per cent of the cases of syphilitic retinitis, 75 per cent of the tubercular keratitis patients and 69 per cent of corneal ulcer patients attended clinic only once. Eighty-one per cent of the cases of glaucoma made three visits or less. None of the cases of retinitis, optic atrophy or purulent conjunctivitis made more than three visits.

Consider the 103 patients from the group of selected cases presenting serious eye diseases. If the examining ophthalmologists spent even five minutes in the examination of their patients, they practically wasted 8 hours of their time, to say nothing of the time wasted by their assistants. With a well conducted social service in a clinic, the number of patients who are examined once and fail to return should be reduced to 20 per cent or less.

The clinic clearly assumes some responsibility for its patients. That such a large proportion of patients presenting eye conditions which may lead to blindness and the accompanying financial disability does not return for treatment, is a stigma of failure on the part of the clinic to assume a responsibility that the administrative and medical authorities should seriously consider.

The records show slight evidence of any attempt to insure proper care to the patient beyond the filling of prescriptions for drugs and glasses, and very little appreciation of the need

to protect other members of society from infection. One socially minded physician recorded the fact, that in the family of a trachoma patient who attended clinic twice, there were two other children infected with the same disease. The record shows no attempt to have been made to bring the other members of the family under care, or to insure the cure of the original case. There was no evidence of any effort to bring tuberculous cases under proper hygienic conditions. In the syphilitic group, laxity was also evident. While it is customary to take Wassermanns, it is by no means a universal practice. The following figures show to some extent the failure of the clinics to take advantage of the Wassermann reactions as a diagnostic procedure in the cases of eye conditions which may be caused by syphilis (iritis, interstitial keratitis, retinitis, etc.) Of the 79 such cases found in this study, only 36 were given this test, and of these, the results were negative in 17, doubtful in one, positive in 10 and not recorded in 8. Further, the records examined failed to show that the clinics brought the patient who showed a positive reaction under arsphenamin treatment. There was evidence in the record of the provision of proper care for only four out of the ten cases for whom a positive Wassermann report was recorded.

As a result of these studies the following recommendations were made by the Section on Ophthalmology of the Associated Out-patient Clinics in regard to follow up work in ophthalmology.

"Systematic follow up to insure continued treatment for at least the following types of cases should be instituted:

Atrophy, optic.
Conjunctivitis, purulent.
Choroiditis, tubercular.
Corneal ulcers.
Glaucoma.
Iritis.
Keratitis, interstitial.
Keratitis, phlyctenular.
Keratitis, tubercular.
Neuritis, retrobulbar.
Ophthalmia, sympathetic.
Papillitis.
Papilledema.
Retinitis, diabetic.
Retinitis pigmentosa.
Retinitis, syphilitic.
Sarcoma of the choroid.
Trachoma.
Uveitis.

"Physicians should be responsible for seeing that the patient is informed of the nature of the trouble and the importance of treatment, and for deciding on what date the patient should return."

"The social service department, with such clerical assistance as is necessary, should note the name of the patient whose return is desired, and thru the proper methods endeavor to secure his return on the date specified."

The clinics which were first studied have recently been restudied, after a lapse of two years since the first survey. Some clinics have seen the advantage of utilizing the social service department for following up cases, but the majority of clinics have as yet no definite follow up policy, utilizing the aid of the social service department only casually. Only a few clinic records show the outcome of cases, indicating, for instance, the proportion of cases who are prescribed for, but do not obtain lenses, or the proportion of patients who fail to carry thru the treatment, particularly in instances of infectious or serious eye diseases, as for example, glaucoma. Also, no record is kept of the patients who come to the clinic once and fail to return.

Special study of 500 case records in August, 1926, in two institutions, by Miss M. K. Taylor of the Associated Outpatient Clinics of New York City, shows the following:

Records are so incomplete as to make it impossible to determine the disposition in 395 out of 500 cases (79%). There was no record of advice or treatment in 105 out of 500 (21%).

Forty-six, or 9%, of the 500 cases fell into the group of 19 selected diseases which may cause blindness. There were 2 cases of keratitis; 6 of trachoma; 6 of iritis; 11 of corneal ulcer; 1 of glaucoma; 2 of optic atrophy; 1 of retinitis; 17 of purulent conjunctivitis. Of these 46 cases 23, or 50%, attended the clinic only once; 7 twice, 8 attended 3-4 times; 5 attended 5-9 times; 2 attended 10-19 times; 1 more than 20 times.

There was no record that the aid of Social Service Departments had been sought to keep these patients under treatment, and little effort was apparently made to insure completion of treatment in other types of cases.

Of the 217 refraction cases, only 113 (52%) actually obtained glasses; 81 (37%) were given drops to put in at home and never returned to the clinic; 6 (3%) were told to return later for drops and never did so; in 17 cases (8%) the refraction was completed and the patient given a prescription, but he failed to secure the glasses.

The waste of professional time is obvious. In addition, there was waste of the patient's time, clinic fees, and in some

cases deposit for glasses, and the economic waste to the community in terms of impaired industrial efficiency because of defective vision.

Of 20 recommendations for hospitalization among these 500 cases, only 11 (55%) were known to have been carried out; 4 (20%) were unknown; 5 (25%) were known not to have been carried out. The Social Service Departments were called on to assist in arranging 3 of these admissions.

Of the 500 cases, only 9 (2%) were on record as being known to the Social Service Department. It is probable that other patients obtained fee remissions thru Social Service, but no record was kept of information secured from the patient.

It is evident, in spite of the original studies and recommendations made for improvements that little use is still made of the Social Service Departments as an aid to keeping patients under care until treatment is completed, and that their assistance is seldom sought, either in connection with interpretation to the patient, while he is in the clinic, or for seeking to discover and remove obstacles to the completion of his treatment.

The question of follow up work is certainly extremely important, as shown by Derby in his pioneer work, and by our observation in the various clinics. "Follow up," in the sense that the patient is allowed to escape and is then pursued, should not be necessary. There should be developed instead a kind of "follow thru" or "case control," which anticipates and prevents the usual causes of incompleted treatmert. We feel that this work must begin with the physician. He must have the details of the history, and should at least appear to take a sympathetic interest in the patient's story. It is frequently impossible, on account of time, for the physician to take a detailed history himself, but this can be done by a sympathetic assistant, and still inspire confidence.

There is no doubt that a routine subjective and objective examination not only gives the physician valuable data, but also makes the patient feel that interest is taken in his case, and that the physician really has sufficient data to make a diagnosis. Much of this examination may be made by trained assistants, who are not necessarily physicians or trained nurses. If the history and examination are adequate, the patients will seldom need the aid of follow up work, even tho the physician does not impress the patient by his personality, interest or sympathy. But there are several other points

which are extremely important and should not be neglected. The first is to impress the patient with the necessity of completing treatment, and to explain the condition in such a way as to allay fear. Most patients fear blindness or some other serious result, and the physician, if possible, should give the patients a few encouraging words, and discussion of the prognosis, or other points which might arouse fear, should be carefully avoided while the patient is present.

In most instances, it is important to give the patient written instructions. This is particularly so in the treatment of glaucoma. The following instructions to glaucoma patients were written by Doctor Eugene Blake:

ADVICE TO PATIENTS WHO HAVE GLAUCOMA.

"You have been told that you have glaucoma—a condition often called hardening of the eyeball. This is essentially a chronic disease, and means that you will always have to take care of your eyes. If you do this and follow certain rules of living, and use the treatment prescribed for you, there is a very good prospects of retaining your sight. If you fail to do this, a gradual loss of sight is certain to follow.

The following rules are suggested for your own benefit, believing that you wish to cooperate with your doctor in securing the best results of treatment of your eyes.

1. Put the drops into your eyes as often as you were advised to. Do not let anything interfere with this—any other sickness in yourself or family, absence from home or any other condition.

2. Do not use belladonna in any form, and if your physician prescribes for any illness, tell him you cannot take bella donna.

3. Avoid alcohol, tobacco and excess in eating.

4. Keep your bowels in good condition.

5. Be sure that your teeth are healthy. An X-ray examination may be necessary.

6. Have your physician examine your urine and take your blood pressure at least once a year.

7. Avoid worry as far as possible.

8. Return for reexamination when requested."

What part does social service play in this new conception of follow up? The functions of hospital social service in gen-

eral are to assist in the medical care of the patient. This assistance is rendered by:

(1) *Interpretation.* This may consist in collecting and reporting to the physician facts pertaining to the personality and environment of the patient which may effect diagnosis. It may consist in interpreting the patient's condition and the physician's advice in lay terms to the patient or to his family, or to anyone else, teacher, employer, nonmedical social worker—who needs to understand. By such interpretation to patient or parent, consent may be won for operation and necessary diagnostic procedures.

(2) *Making adjustments which will make treatment possible and overcoming obstacles.* For example, we may mention the helping of needy patients to obtain the medicines they require, lenses prescribed, protheses and other things required for the proper treatment of the case, for which the patient is unable to pay, also free beds, operating room service and nursing. The social service department may make arrangements for the patient to appear at the hospital at the proper time, as it has been our experience that several patients have become blind, particularly glaucoma cases, by not having been taken to the clinic at the proper time, either because there was no one to conduct them there or because, in the absence of the social service, the family had not been instructed as to the importance of treatment and the risk involved in neglect. In certain cases, home treatment is made possible and carried out thru social service aid.

(3) *Utilization of community resources in arranging care of patients.* This may involve the bringing into the home of other organizations—nursing, welfare, etc. It may involve reference of patients who are blind or have reduced vision to proper organizations for their care. Patients who have become blind should be placed in touch with agencies equipped to teach them and secure employment for them. Patients with defective vision at the school age should be followed to see whether it is necessary for them to be placed in sight conservation classes.

In addition to these principal functions of social service, the social worker may be of great use in the management of the patient in the clinic, by directing him thruout his course of treatment in clinic and hospital, assisting him to understand procedures and directions, and perhaps aiding in clinic demonstration of directions.

The social service department may also play a part in research and teaching. It would seem as tho it were fitting for this department to aid the physician to obtain special medical, as well as social data in selected groups of cases, and to aid in the preparation of scientific papers which should endeavor to stress the social side of medicine, for in this way our physicians will be educated as to the value of social service and follow up work. By following cases and making it possible to complete records, valuable data is made available for teaching physicians as well as social service workers, and aids the physician to obtain certain groups of patients for demonstration and study. By studies of groups of patients, valuable facts may be discovered which have important bearing on public health.

Organization of Clinic Which May Help to Insure Completion of Treatment.

We believe that each physician should have his own patients, and that if he tells the patient that he personally wishes to see him at a certain time to do some definite thing, the patient will seldom fail to keep the appointment.

Appointments should be recorded, as this will also act as a check of attendance and form the basis for the follow up system. If the condition is not serious, the patient's name may be recorded on the same day a week later, and if he fails to appear on this date, a card may be sent. If the condition is serious, a card should be sent the day of the patient's expected return. To facilitate this work, and for other reasons, the patient's address and telephone number should be recorded on the clinic record.

Summary of Essentials of a Follow Up System.

a. Impress the patient with the desirability and importance of returning to the clinic, by courteous treatment, a thoro examination, carefully explained and written instructions, and by making a definite appointment for his return. The time for return should be decided by the physician, and note made on the record as to the disposition of the case.

b. A card index or appointment book should be used to note the patient's name and what he is returning for. A card or letter should be written or personal visit made, according to

the physician's directions. If it is impractical to follow all cases, a specific group should be selected, as completed thoro work on a few cases will do more to raise clinic standards than incompleted superficial studies of many cases.

c. The results of follow up studies should be reported monthly at the clinical conferences, and should show the number of patients treated and their disposition.

The following outline may serve as a guide to the social service worker and ophthalmologist in selecting cases for special attention.

a. Orbit; tumors and infections which may lead to blindness or death should be carefully followed.

b. Glaucoma; patients with glaucoma should be particularly watched, and this can best be done by the active cooperation of the social service department. If these patients are allowed to go even two or three days without proper treatment, it may mean loss of the sight of an eye. Of the glaucoma group first studied, 81% made three visits or less.

c. Optic Tracts; patients with lesions which effect the optic tract, for example, intracranial tumors, and particularly diseases of the pituitary body.

d. Eyelids; ptosis, particularly congenital, and in these cases a study of the family is also indicated. Tumors of the eyelids.

e. Muscles; patients who have paralyses of the muscles of unknown origin, and particularly patients with syphilitic paralyses, should be carefully studied and kept under observation. Children with strabismus, who are supposed to wear glasses and to report for observation and fusion training, should also be followed up.

f. Conjunctiva; gonorrheal infections, syphilitic infections, trachoma and keratoconjunctivitis eczematosa should be followed and studies made of the family and living conditions. Purulent conditions should be carefully followed, as it is our experience that unless this is done, 83% may attend only once, and no patient studied makes more than three visits.

g. Cornea; patients with ulcer of the cornea should return for observation and treatment, for neglect may mean the loss of an eye. In the first survey, it was found that 69% of the corneal ulcer patients attended the clinics only once. Malignant growths of the conjunctiva and cornea should also be kept under observation. Patients with tuberculous keratitis

and interstitial keratitis due to syphilis should report regularly for observation and treatment. Only one visit was made by 75% of the patients with tuberculous keratitis. These two groups, or even one of these groups, would require the time of a full time worker in a large eye clinic, if proper attention were paid to the family and personal life and relations and these patients.

h. Iris; patients who are suspected of having tumor of the iris should be under careful observation. Those with iritis should be followed to see that they have studies made of their blood, teeth, nose and throat, gastrointestinal tract and genitourinary tract, in the attempt to determine the etiology. Syphilitic patients should be carefully followed for a long time, and tuberculous patients should have hygienic treatment.

i. Lens; patients with cataract, particularly of the congenital or hereditary type, should have studies made of their families, and should be warned against the danger of transmitting cataract to the offspring. Those with incipient cataract should be followed up to see if they are continuing treatment, and to make sure that they are having studies of their general conditions made to determine any possible etiologic factor.

j. Uveal Tract Diseases; sympathetic ophthalmia cases should report regularly for observation. Patients who are suspected of having tumor of the choroid, or actually have a tumor of the choroid or ciliary body, should certainly be followed. Those with inflammation of the uveal tract should be followed to see that they continue treatment, and that they have studies made to determine the etiology of the inflammation.

k. Retina; children with tumor or suspected of having a tumor should be particularly watched, both before and after any operative procedure, and other children in the family should be observed. Patients with retinal hemorrhages should be followed to see that they have etiologic studies made, and patients with diabetic and socalled albuminuric retinitis should be carefully observed, and the effort made to obtain general medical treatment for them. Patients with syphilitic infections of the retina should be treated by the department for the treatment of syphilis, and should be carefully followed, as it was found that 80% of the patients with syphilitic retinitis attended the clinic only once. Patients with

retinitis pigmentosa and Tay-Sachs' disease should be studied in regard to their family history. Patients who have intraocular foreign bodies should be followed to see that they report for proper treatment and study of the other eye, to see that sympathetic ophthalmia does not develop.

l. Optic Nerve; patients with optic atrophy, particularly if due to syphilis, should be followed, and they should report for treatment in the department of syphilis. Patients with papillitis should be carefully studied for the cause, and follow up given to see that they return for etiologic studies. Patients with papilledema should be followed to see that they report for etiologic study, as most of these patients have brain tumor as an etiologic factor. They should also report for repeated field studies, studies of their vision and blind spots. Patients with retrobulbar neuritis should be followed to see that the proper etiologic study is made, and particularly that the proper studies are carried out in the departments of rhinology and roentgenology.

m. Injuries and Industrial Accidents; many problems arise in the work in which the active cooperation of the social service department is of the greatest importance. The patients must be cared for and followed up, his employer must be consulted, and the family must be provided for. Some of these patients at first refuse to enter the hospital, and they should be followed to protect them from their own ignorance.

n. Refractive Errors; patients with high degrees of error frequently need telescopic lenses, and these can be purchased by most patients only thru the social service department. These patients should be followed to see that they can use their lenses, and are helped to find suitable employment. They must also be followed to see that what little vision remains is preserved. Myopic patients, particularly when there is a tendency to rapid progress of the condition, should be systematically followed, and measures instituted to improve their general health and living conditions. Patients with conical cornea need general and repeated eye examination, and should be referred to the social service department.

o. *Follow up of operative cases both before and after operation.* Before operation, to see that the proper preparation and etiologic studies are carried out, and after operation, to see that the patient reports at the proper intervals for restudy, is an extremely important function, and should be carried out by the social service department, by having every patient who

is to be operated on report both before and after operation. The patient should report the first clinic day at the end of one, three, six and twelve months, and each succeeding year.

From the recent studies of 47 eye clinics in Manhattan, Bronx, Brooklyn and Queens, made by Miss Louise C. Bentley of the Associated Out-patient clinics of New York Tuberculosis and Health Association, it would seem, first, that more social workers were needed; second, that the physician should be instructed in the use of the social service department; third, that more follow up work is needed to do three things: first, to assure the proper treatment for the patient; second, to conserve the physician's time and make data recorded available and valuable for research and teaching; third, and probably the most important, that blindness may be prevented.

The following is a brief summary of the findings:

Social service departments are available for the use of 31 of the 47 eye clinics studied, and yet the ophthalmologists avail themselves infrequently of the service offered.

In two, a social service worker is present at each session, and in one clinic, a nurse with social service experience does this work, and special stress is placed upon the family health service. No social service is available for 13 clinics, either because the institution does not have a social service department, or because this department is too busy to provide service for the ophthalmologic clinic. Even when service is available, it is reported that "a number of the clinic chiefs seem to consider that the sole function of the social service department is to deal with financial investigation, for the purpose of determining whether the patient is entitled to free glasses." They do not utilize the social service department to supplement medical treatment. The eye cases that particularly need social service are frequently neglected. Upon the physician rests the responsibility for recognizing, in his clinic, the need for adequate aid, and for placing a definite request with the administrator. The physicians, themselves, should recognize that their time can be conserved, and fuller service given to their patients, not only if they are freed from routine clinical duties, but also if they are supplied with the necessary nursing and social services.

Follow Up.

The value of follow up work to the physician and the patients is apparently appreciated in only three of the 47 eye

clinics. In one clinic, all patients who break appointments are followed by letter, and if necessary, by home visiting. In another, the nurse with social service training makes visits, and in a third, where there is a full time social worker, selected cases are followed after conference with the physician.

It is urged, in conclusion, that "more eye clinics should observe the principles involved in the follow up of incompleted treatment of the patients. Unfinished treatment is of little value to the patient and unsatisfactory to the physician. Follow up begins with the reception of the patient in the clinic, and the results depend upon his mental and physical reaction to the treatment he receives. The mechanics of follow up, sending letters and making home visits, should be considered secondary in this broader conception of case control."

We believe that the studies made of ophthalmologic clinics and records in New York City and Brooklyn justify the following conclusions:

(1). Social service departments in relation to ophthalmologic clinics serve many purposes. They secure the proper treatment for the patient; they see that those who have defective vision or are blind are referred to the proper organization for care; they prevent blindness by seeing that the patients are followed up and report at proper intervals for observation and treatment; they conserve the physician's time and energy, and make his work valuable for research and teaching, by seeing that his instructions in regard to etiologic studies and treatment are carried out until the case is discharged. This is of prime importance in view of the fact, that in a selected group of 193 patients with serious eye diseases, who should have returned for observation to three of our leading New York ophthalmic hospitals, 103 attended only once.

(2). The survey of eye clinics made in 1923 and partially repeated in 1925, shows that many of the hospitals and clinics and some of the physicians are not yet alive to the necessity for having social service and follow up work.

(3). In the hospitals that have social service departments, the ophthalmologic clinic did not always take full advantages of the services rendered. This defect should and can be remedied by the education of the physician and the presence of the social service worker in the clinic.

(4). Follow up work must begin with the physician, for unless the patients are impressed by the physician's skill and

personality, it will be difficult or impossible to get them to return. Definite appointments for the patients with a certain physician at a definite time, will minimize the necessity for follow up work and will act as an index if checked against daily attendance. The value of careful explanations by the physician to allay fear and yet impress the patient with the need for treatment, is greatly enhanced by written directions and by the sympathetic attention of the social service worker, and will prove an important factor in eliminating the need for follow up work.

(5). When ophthalmologists fully realize how valuable social service and follow up work are in the conservation of medical effort and the advancement of the science of ophthalmology, and demand this service, the directors of our hospitals and ophthalmologic clinics will see that this invaluable service is made available for their use.

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DISCUSSION.

Dr. W. H. Crisp, Denver: Few of us are likely to dispute the desirability of the proposals offered by Dr. Berens in his most conscientious and laborious investigation. But many years will probably roll by before the glaring deficiencies which he has revealed are made good thruout the country. There must be reasons why clinic patients do not return for completion of their treatment. In some cases, perhaps, they decide to consult a private practitioner; in other cases, it may be that the treatment is carried on more or less faithfully according to directions given in the clinic, altho the patient does not return for continued observation. A third reason may be the quality of the medical service afforded in the clinic.

Much excellent work is done in outpatient departments all over the world. Yet in visiting various clinics, I have often had the feeling that the work done in them was sadly inferior to that done in private practice. There are reasons for this. The time for clinical practice is often grudgingly spared by the physician. In private practice, the physician is the professional servant of the patient, and his services may be terminated at any time if they are regarded as unsatisfactory. Such termination of employment involves reduction of income. In the outpatient clinic, to speak frankly, the average physician does not serve mainly for altruistic reasons, but for selfish ones. The clinic tends to keep him before the profession and the public as an expert in his line. But he is most interested in cases of an unusual character, and especially cases involving major surgical operations. Even such cases in outpatient work are apt to be viewed with enthusiasm in their earlier, and with weariness in their later stages.

In the clinic, the physician is not in any very serious degree subject to discharge if his services are unsatisfactory. The question arises whether such liability is not just as desirable here as in private practice, but it is hard to maintain such a liability with regard to one whose services are entirely gratuitous. Personally, I feel that it should be possible to hold the physician in an outpatient department just as strictly responsible as is the instructor in an academic educational institution, for example. The only basis upon which this can very well be accomplished is that of payment of the clinician as a part time servant of the institution, altho it goes without saying that such payment can hardly be commensurate with the financial rewards of private practice. The institution should then lay down reasonably precise rules for the management of the clinic, including the details of follow up work suggested by Dr. Berens, and should maintain a reasonable spirit of discipline in its staff.

Dr. W. H. Wilder, Chicago: I think there can be no dissent from the proposition that the social service worker has been of very great benefit in the clinics of large cities. This movement has demonstrated its usefulness to such an extent, that it would be extremely difficult to abandon it. We have seen the operation of it for a good many years in the clinic with which I am associated, and our conclusions there would certainly bear out those which have been presented to you by Dr. Berens. The cases are followed up (or followed thru, which might be a better term), they return more promptly to the clinic, they get better service, and we probably accomplish better results.

There is one thing, however, that has struck us, and I suppose Dr. Berens and the men in New York have observed the same thing, and that is the increasing expense to the clinics from the employment of these social workers. They are coming to have a rather high opinion of their services, and the expense is increasing greatly, so that the suggestion has been made, and I think it has a good deal of merit, that we ought to have our medical students engage in some of the duties of the social service worker, and the follow thru work might be very appropriately done by some of the students who are attending these clinics. This plan might be made feasible by having regular assignments made to medical students to do some of this follow up work.

I am struck with the value of the suggestion made by Dr. Berens as to printed instructions for the patient in regard to the follow up treatment. Such instructions, carried out by the social service worker, either directly or in the way of impressing the patient with the great importance of it, will be extremely valuable. How frequently we find that a young and inexperienced assistant in the clinic will not give definite and accurate enough instructions to the patient as to just what he is to do in a given case. Even in a simple matter like putting drops in the eye, the patient may come back, especially if he has been given such a drug as mercurochrome, with his face painted like a Comanche Indian, because, from lack of proper instructions, he has not been able to get the drops in his eyes, but has smeared them all over his face. Many patients, when they open their eyes, open their mouths too, and if they are given a strong drug like atropin without proper instruction, they are quite likely to get more of it in their stomachs than in their eyes. I have seen more than one case of atropin poisoning from such neglect.

DR. ANDREW W. MCALESTER, JR., Kansas City, Mo.: I want to call your attention to one thing that I think ought to be taken into account in this social service, and that is the enormous number of clinics in New York City. That is rather appalling. Why so many clinics?

Another thing is the large number of people who come to these clinics for free service when they should pay. The other day I went down to our own outpatient department, and two traveling men came in to get a shot, and we found they were two of the highest paid salesmen in Kansas City. We have had patients come to this department for free treatment who come in taxicabs. These social service workers have a duty to the doctor, and that is to see that these clinics do not pauperize the doctor. This is an important function of the social worker—to protect the doctor and stop this pauperization of the medical profession.

DR. ANNA B. LEFLER, Los Angeles, Calif.: It has been stated that only 10 per cent of the physical examined of the public schools follow the corrective work advised.

The waste occurring in the clinics as reported by Dr. Berens, added to the failure of the schools to profit from their medical examinations makes the total loss appalling.

Dr. Berens has brought up a timely subject.

DR. WM. FOWLER, Detroit: It seems to me the conclusions that Dr. Berens has brought out are very excellent and ought to be followed everywhere if it were possible. The great trouble, as I have seen it, not alone in the hospitals in which I work, but in other hospitals in Detroit, New York and Chicago and everywhere else in the United States, and Europe as well, is because all of these clinics are run on the production basis rather than on a scientific basis. We will never be able to get the necessary help from social service, from the nurses and from the others engaged in the work, until we in some way or other can find a suitable working basis between the two great fundamentals governing the clinics that are so diametrically opposed to each other. First, there is the very great desirability in some places of a clinic having the reputation of having a very large number of patients. Every hospital that is doing free work wants that reputation. Against that, and directly opposed to it, is the very great desirability on the part of the community to have no free clinics whatever. If you can get these two ideas, these two fundamental objects, these two principles, pulling together in some way, we will have solved the great question of free clinics.

I believe, however, as has been suggested, if we would, instead of trying to clean up all the patients that are waiting for us, take two or three and forget the others and do those thoroly and carefully, using all the means at our command—social service and other offices of the clinic—that we will in time be able to cut down the number of cases that we have taken care of, but we will have taken care of those we do treat very much better.

DR. PETER T. GRANT, Grand Rapids, Mich.: I have been serving for the past year on a committee in connection with social welfare work in the city of Grand Rapids, and I think, as the result of my experience in that position, I ought to sound a word of warning in regard to the management of social welfare work among the sick, and to emphasize that this

should be under control of physicians connected with the welfare organization. We have a strong social welfare organization, but last year the Kent County Medical Society found that many patients were being served free who were well able to pay. This welfare organization should investigate carefully the financial condition of all cases. Our welfare organization is entirely maintained by voluntary contributions of the people. The doctors have contributed their services free of charge, but we found there were three points of injustice involved in this work. The first is that the money contributed was being used for certain persons who were well able to pay for the service. Second, the doctors by contributing their services free to these patients deprived private physicians of their legitimate practice and income for that service. Third, we found this condition was acting badly on the recipient, psychologically, because it stimulated still further the demand for free service among people who were not entitled to it. These same people will hold a higher respect for those who render them this service where proper remuneration is given.

Dr. Conrad Berens, New York City, (closing): I thoroly agree with Doctor Crisp that it is quite important to pay the men who work in clinics. As a matter of fact, this was recommended in New York four years ago, and for two or three years some of the hospitals have paid some of their assistants. Within the past year, the Manhattan Eye and Ear Hospital has paid some of the ophthalmologists to do refraction.

Doctor Wilder's suggestion in regard to having medical students do the follow up work is an excellent one. They woul dlearn a great deal from this type of work if they would study selected groups of cases and publish the results of their investigations.

The abuse of clinics has been referred to, and it is unfortunate and unpardonable, particularly in New York. The suggestions made by several of the men who have joined the discussion would do a great deal towards overcoming these abuses. The social service departments in the New York hospitals are as a rule loath to look into the financial status of the individuals, but it seems to me, that in selected cases, this should be one of the functions of these departments.

BAND KERATITIS.

ARTHUR J. BEDELL, M.D., F.A.C.S.

ALBANY, N. Y.

The deposition of calcareous salts in Bowman's membrane is common. When the deposit is in the form of a ribbon, it is called "Band Keratitis," and is observed in two forms; the primary, which is rare, and the secondary, which is frequently seen.

Few articles have appeared in English literature since the disease was first described by Dixon, in 1848, as "a curious form of opacity depending upon spontaneous deposit of earthy salts between the epithelium and the anterior surface of the cornea," and referred to by Bowman in the following year as "a symmetrical opacity of both corneae, extending horizontally over the central region and obstructing vision; consisting of an earthy deposit limited to the anterior elastic lamina." He further stated, "I am not aware that these are particularly described in books, nor whether they admit of removal or even arrest. They are probably connected with an imperfect nutrition of the eyeball, and must be left to take their course."

Graefe, in 1869, described band keratitis in the discussion of the pathology and treatment of glaucoma. This was followed by many papers, including those of Nettleship, in 1879; Leber, in 1896; Usher, in 1890; and Suker in 1900.

One type of this disease, the secondary, follows chronic iridocyclitis, advanced glaucoma or intraocular affections as a result of which the patient has lost sight. The other form is primary and not associated with any other ocular disease. It is well to have a clear conception of this clinical entity, and not include every palpebral fissure opacity as band keratitis. Suker discussed the disease and proposed the name "Keratitis Petrificans" as a distinctive, comprehensive term. Deposits may follow an inflammation of the cornea, and are usually between Bowman's membrane and the external limiting layer. The typical opacity varies in color from silvery-white to gray-brown. One author has described it "as appearing like dirty

ground glass viewed from the unground side." It is a resistant layer like the glaze on china in appearance and feel.

When this corneal opacity is associated with an intraocular infection, it is secondary to it. The difference between secondary and primary band keratitis is based on the history and the physical examination of the parts affected, including the slit lamp procedures.

The secondary bands are well illustrated by two patients, partial in the case of H. H., a twenty-five year old male, who came to have a cataract removed. For three years, his left eye had been sore, and for two years blind. In addition to a secondary gray cataract, he had several isolated areas of

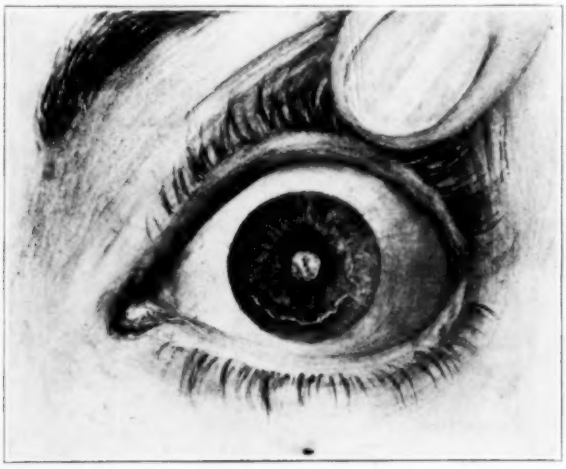

Fig. 1. Partial Band Keratitis. Early stage of formation.

corneal opacity. Four years later, 1926, the opacity had increased so as to suggest the distinct lines illustrated in the drawing. There was a wavy line at the lower palpebral fissure, from which there was an upward extension on the temporal side. Under the slit lamp, the epithelial surface over the infiltration was smooth. Directly beneath it was a whitish mass, consisting of a thin layer in which there were many rounded, black appearing areas, the characteristic appearance of the disease under discussion.

The complete band is well shown in one reported at the American Ophthalmological Society, in 1925. This was a case of absolute glaucoma in a sixty year old woman, who had been blind for several years. There was an ovoid zone of gray infil-

tration roughly corresponding to the palpebral fissure. With the slit lamp, the vessels at the limbus were distended, with loops which extended into the superficial cornea above and to the outer side. There was nothing noteworthy in regard to the blood vessel walls. The entire corneal surface was stippled. In the oval area of infiltration, the two grossly visible bullae were seen as a collection of fluid in the epithelial layer. One was somewhat quadrate, with five irregularly wavy elevated epithelial line extensions; the other, a cord of epithelial elevation with most of the fluid collected in an oblong space, altho there were traces of it in the raised tube which extended from each end of the bulla.

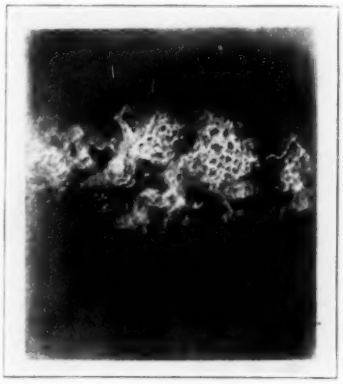

Fig. 2. Typical infiltration as seen under the slit lamp.

Beneath the epithelium and Bowman's membrane were two definite kinds of infiltration; the more anterior area was white, and suggested a thin calcification layer. The meshes were large and the framework flat. The effect was that of holes punched out of white paper. The other opacity was deeper and had a wavy outline, like filamentous threads, which extended into the various layers of the stroma. These changes were similar to those usually found in any type of band keratitis, whether a narrow arc at the limbus, or a definite ribbon occupying a major portion of the cornea.

The primary form occurs, as stated, in an otherwise healthy eye, usually in elderly people, and most frequently in those exposed to irritation, such as dust or certain chemicals. It is characterized by a progressive extension of opacity, most often beginning as small flakes near but not at

the limbus, in the palpebral fissure. Occasionally, the opacity may be seen as an isolated speck. It is essential to remember that the process may never go on to the formation of a distinct band, but may remain as a more or less isolated arc.

Mrs. S. T. complained of a progressive decrease in vision for the last twelve years, with lacrimation for the past three weeks. Vision of the right eye was 20/50, and there was an irregular band of lace like opacity which extended across the cornea. The left eye, vision 5/200, with a similar but denser band, which extended from about one-quarter of a mm. inside

Fig. 3. Bilateral band showing its typical location and characteristic gross structure. (a) The stippling as seen in the indirect light. (b) The infiltrated zone with the characteristic rounded, black appearing areas. (c) The fine fibrillations on the posterior surface of the infiltration.

the limbus across the cornea, with its widest portion over the pupillary area. It consisted of many gray and white layers with several small distinct black holes, some of which were round, others oval. As there was no other pathologic change in either eye, she was advised to have the film removed. No one whom she consulted would agree to the operation and, altho first examined in 1922, she has, as far as we know, had no operative intervention.

Her lack of courage was contrasted by the experience of Mr. W. H., a strong, vigorous, retired New York police captain,

seventy-one years of age, who came in after having seen "a lot of doctors." Among the list were many of experience and great skill, some of whom said they had never seen his kind of an eye, and one said nothing could be done to materially help his sight. He was first examined on the 9th of April, 1925. Right eye, vision 6/200. He had a broad band of gray opacity extending across the cornea, with the exception of a narrow clear zone between it and the limbus. With his partly closed eyelids, this band, 4.8 mm., wide occupied the exposed portion of the cornea. This was widest in the periphery, with a convex margin toward the apex, corresponding to the shape of the eyelid when it was in apposition to the cornea. The lower margin was also curved like the lower lid. There was nothing abnormal in any other part of the eye. Pupil 3.5 mm., responding promptly to light. The iris was a good color, the pupillary area back of the iris clear. The gross appearance of this band was that of a network of grayish fibers with spaces which looked black, because of the contrasting absence of infiltration. These comparatively clear areas were oval and round, conveying a definite impression of holes punched out of a gray sheet.

Left eye, vision 6/200. The condition of the eye was practically the same as the right. A grayish band similar to that on the right extended across the cornea.

Under the slit lamp, in indirect light, there was a definite stippling of the epithelial surface. There was a continuous sheet of opacity, with several rounded, pigmented masses anterior to the network. The infiltrated zone was anterior to the corneal nerves. With direct light, the network was definite with irregular channels. At the junction of the lines of infiltration, there appeared black dots, which were in reality clear areas. There were also many rounded and oval black appearing spots in this gray sheet, over which the epithelial layer was distinctly hazed, indicating that the infiltration was probably deeper than Bowman's membrane and some of it was in the superficial stroma. On the other hand, the clear areas may have been uninvolved Bowman's membrane.

On the 5th of November, 1925, he entered the Albany Hospital, and the right eye was operated upon. There was practically no reaction. Four days later, November 9th, the left eye was operated upon. He left the hospital on the 15th of November, and when last seen on the 8th of January, 1926,

the vision of the right eye was 20/30 and Jaeger 1. Vision of the left eye, 20/30 and Jaeger 1. With the slit lamp, there was an irregular edge where the fragile layer had been cracked off of the remaining sheet. The corneal epithelium had reformed, leaving a clear glistening surface. The eyes were quiet. By letter, in August, he reported complete functional recovery.

OPERATION: The patient enters the hospital the night before. The eyes are washed with boric acid solution and a drop

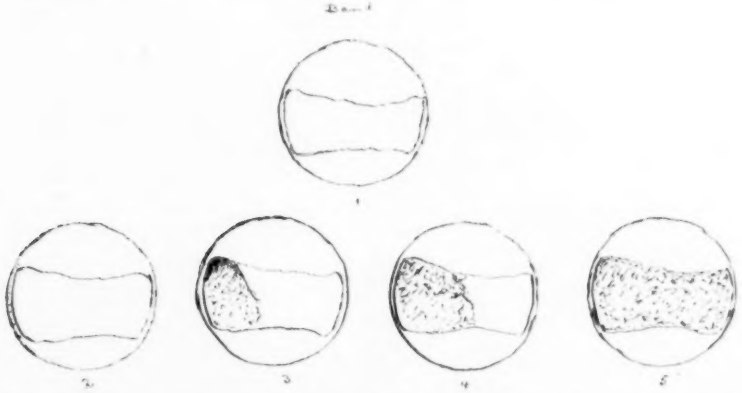

Fig. 4. A schematic representation of the detail of operation. 1. The outline of the band. 2. Primary incision thru Bowman's membrane illustrated by dots. If the calcareous layer is too resistant, a small piece is cracked loose. By rocking the knife from side to side, it is easier to free the layer and proceed with the dissection. 3. The first mass lifted by the spatula. 4. The advanced stage of the operation. 5. The band completely removed.

of 25% argyrol instilled. In the morning he is given the same preparation, and when he arrives in the operating room, one drop of 4% cocain is dropped over the cornea every two minutes for six drops. The eyelids are scrubbed with green soap, the eye thoroly flushed with boric solution. An ordinary lid speculum is placed and the eyeball, held firmly with strong fixation forceps, is turned to the nasal side. A very sharp, small scalpel is used to start the dissection, which should be begun near the temporal limbus, preferably just to the outer side of the calcified zone. The first incision should go thru Bowman's membrane. If the film is too hard and the knife only scratches its surface, it will be necessary to crack a small piece off. Then, in the partially clear area, a thin silver spatula or a special curette is steadily but gently forced between the calci-

fied layer and the clear underlying cornea. If the fragile layer cracks, it is only necessary to carefully insinuate the spatula beneath the broken edge and lift the shell from its base. The layer is almost certain to break, so that the edges are irregular, but by using sufficient well applied force, the entire band may be removed. The essential to success is the clean removal of the opaque layer without injury to the underlying clear cornea. If, after the major portion of the film has been removed, small gray specks remain in the pupillary region, they should be lifted off.

Following the operation the eye is washed with boric solution, atropin instilled and a compress bandage applied. Healing is uneventful. There is little or no injection of the globe, no pain and usually no discomfort.

The eye is irrigated once a day and the dressing reapplied. If the stroma has not been damaged, healing is complete in a few days and glasses may be adjusted within three weeks, and the patient may use his eye without restriction.

Thru an unfortunate circumstance, we are unable to present the section of the material removed. The specimen was taken to the laboratory, stained and examined, but found to be so thick that good photomicrographs could not be made, and in the attempt to make the section thinner, the whole specimen was destroyed. Dr. Jacobson's report follows:

"MC:—A piece of white membrane, irregularly triangular in shape, about 1x.3 cm. in size and of paper thinness. It is too thick for examination by transmitted light unstained, and too thin for ordinary histological sectioning. It is fixed in 50% alcohol and stained with haemotoxylin and eosin. This shows it to consist of a mosaic of flat epithelial cells, the layer being about three cells thick and supported by a delicate fibrous membrane. The individual cells are separated by a narrow zone of intercellular substance, which stains blue with haemotoxylin, and which may indicate the presence of soluble calcium salts. No insoluble calcium salts present.

In trying to remember the specimen, it broke into many fine pieces, which prevented further study."

In 1909, Wiener presented before the Section of Ophthalmology at the American Medical Association, a paper on "Regeneration of the Cornea," at which time he referred to his work on rabbits and described his operation for the removal of corneal opacities. It was my good fortune to see many of

his animals, and to note how wonderfully well they had healed and how little corneal cloudiness remained. It was the observation of his work that led me to adopt the present method of removing these calcified sheets.

It is interesting at this time to report the case of Mrs. C. F., forty years of age, who was first examined July 26, 1924, at which time there were several small, isolated corneal spots from which the corneal epithelium was absent. About a year later, the epithelium was again separated from the same areas, and some months later she came in with a large bulla. In October, 1925, several fine specks were located with the slit lamp in Bowman's membrane. By December 29, 1925, they had so increased in size that they appeared as real flakes, irregular in size and outline, with the characteristics of band keratitis.

It is to be recalled that Vogt described a horizontal line of altered refraction in Bowman's membrane. This was seen only with the slit lamp and found especially in people exposed to inclement weather.

Such observations may eventually assist in the solution of the problem now confronting us, the cause of the deposit, which chemists and pathologists claim is phosphat and carbonat of lime. Pathologists are all agreed that the basic lesion is a calcification of Bowman's membrane.

There is, however, no agreement concerning the cause or the treatment of the condition.

Dixon, in his original paper, clearly demonstrated the value of operation. His good example has not often been followed either by the writers of our text books or by clinicians. Beard states that scraping the cornea was practiced long before Galen's time. The modern text books fail to convey the proper impression of either the symptoms of this disease, its prognosis or its treatment. Some authors suggest an iridectomy. The largest and most often consulted work, the American Encyclopedia of Ophthalmology, states, "So far as treatment is concerned, very little can be expected."

It is instructive to read the appendix in Mr. Bowman's work, in which the clinical picture of the disease is well described.

The first report of the disease came from England. So did the last one that we found, which was that cited by Mr. Hudson before the Section of Ophthalmology, Royal Society

of Medicine, February 9, 1923, under the title, "Two Cases of Primary Band Shaped Opacity of Both Corneae." In one, following scraping of the cornea, the vision of the right eye had been improved from 6/36 to 6/24, and the left from 6/60 to 6/12. The other one had not been operated upon. He found the ordinary sharp corneal spud to be the instrument of choice. His impression was that the opacity was in Bowman's membrane.

From a study of our cases and from a review of the literature, we are unable to make a statement regarding the causative factor of this disease. Clinically, it seems certain that mere exposure to wind, dust or irritation is not sufficient to produce it, for otherwise it would be much more common, and the reduced vision resulting therefrom would be much more often observed.

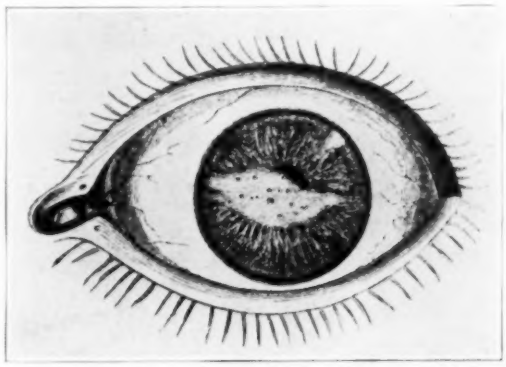

Fig. 5. Bowman's case before operation.

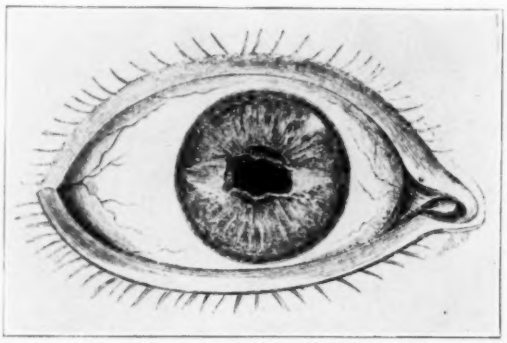

Fig. 6. Bowman's case after operation.

Summary.

Band keratitis is primary or secondary.

When primary, operation is indicated. When secondary, it is part of the general degeneration of the eyeball and does not respond to local treatment.

The diagnosis is made on the appearance of a gray band in the palpebral fissure, with the presence of what seems to be black holes in the sheet.

The differentiation of type is made after the complete examination of the eye.

If there is no pathologic change other than that in the cornea, it is primary band keratitis.

The pathologic change is in Bowman's membrane.

The slit lamp confirms the location of the lesion.

Conclusions.

Primary band keratitis, an hitherto neglected corneal disease, should be operated upon, and the operation should consist of a corneal dissection following the lines here suggested.

The primary and secondary forms must never be confused, and a patient suffering from secondary changes should never be subjected to a corneal operation.

We may best close with Bowman's words, printed in 1849, after he had described his case, "The issue of my case I am anxious should be known, because there are probably many similar ones scattered over the country given up as incurable, and yet within the reach of this method of relief."

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DISCUSSION.

Dr. Robert von der Heydt, Chicago: I will limit my remarks to slit lamp observation on related conditions:

Primary band shaped keratitis is a very rare lesion. I have not seen a typical case in six years of daily use of the slit lamp.

An electric short circuit flash insult of the cornea would probably be an analagous acute lesion. Here we have an epithelial disturbance of the same corneal zone. There is photophobia and a staining of very minute eroded epithelial areas. In mild cases, the photophobia responds immediately to cocain, hence is not of retinal origin.

The epithelial dystrophy of Fuchs probably presents a more chronic type of a lesion somewhat similar to primary band shaped keratitis. In this very rare condition, exposure probably is not a factor, as the whole of the corneal surface is involved.

According to Vogt, this disease is supposed to originate in the endothelium. For some time I was in doubt as to how he could ascertain this with the slit lamp. The cases that I saw extended to the limbus, hence presented no edge or beginning involvement. However, I later found a mild case in which I could see a very irregular posterior corneal surface thru the involved epithelium.

The Staehli line in normal cornea is also an analagous condition. This is a horizontal, more or less pigmented fracture in Bowman's membrane, due to senility. Bowman's membrane is not a membrane in the sense of having two definite limitations, as we find them in Descemet's.

The corneal stroma is condensed anteriorly and presents an abrupt glazed limitation. This is the socalled Bowman's membrane. It has no posterior limitation, for it merges into the stromal structure.

These three conditions are all somewhat analagous to primary band shaped keratitis.

I congratulate Dr. Bedell on his results in operating the case he reported, and the marked improvement he obtained in visual acuity.

Dr. George W. Jean, Santa Barbara, Calif.: I wish to compliment Dr. Bedell on his fearless surgery in these cases, and his wonderful results. Of course an eye with vision of 6/200, due to corneal opacification, is open to fearless surgery.

A point that always worried me, was why the opacity in zonular opacity of the cornea did not go to the limbus. I was in hopes Dr. Bedell would tell us that, but he did not. The reason is this, as Wintersteiner taught me. The opacity is due to lime deposits in Bowman's membrane, and Bowman's membrane does not go quite to the limbus; therefore, you get this little open space of transparent cornea between your opacity and your limbus. This was shown in all the pictures except the ones from Dr. Weeks' textbook, which was evidently taken from Axenfeld's textbook, where it is also misleading.

One other point: Excision of Bowman's membrane is an excellent way to get rid of that lime, but I believe there is another way, and that is by the persistent use of a solution of ammonium tartrat. Fuchs' textbook mentions this, and years ago, at the Eye and Ear Infirmary in New York, I tried it on a case of lime burn. It burned intensely, and the patient did not return. Dr. Otto Barkan has found that you can use a weak solution of ammonium tartrat over a long period of time, and that you will get excellent, nearly miraculous, results in cases of lime burns.

Dr. W. H. Wilder, Chicago: I should like to ask Dr. Bedell how long he has had these cases under observation since the operation, and whether in his opinion there is any likelihood of recurrence of the deposits of lime salts in the remaining scar tissue.

Dr. Arthur J. Bedell, Albany, N. Y. (closing): The object of the communication was to call your attention to primary band keratitis.

Answering Dr. Jean, it will be noted in the paper, that we did the second eye four days after the operation. Eight days after the first operation, the patient left the hospital, and within three weeks had returned to close application, a thing he had not done for many years. This should appeal economically to most of our patients.

Dr. Von der Heydt speaks of the impossibility of getting a clear conception of band keratitis from secondary cases. I am sure, that if you watch, particularly in chronic iridocyclitis or chronic glaucoma, you will find the typical lesions. These extend across the pupillary area like a band. Furthermore, in a blind and painful eye with a well developed band, you can observe thruout the entire field characteristic punched out holes in the dense grey sheet.

Answering Dr. Wilder, the patient has been under observation only since the 5th day of November. The case originally reported by Dr. Dixon retained his vision without change, and there was no increase in the deposit. The one cited by Bowman showed no change a year later. At no time did Bowman refer to a new deposit.

Under the most careful slit lamp examination, we find that the border of the layer, from which the film was cracked, is exactly as it was immediately after the operation.

FRACTURE INVOLVING THE APEX OF THE ORBIT, WITH LITTLE OR NO EXTERNAL EVIDENCE OF TRAUMA, RESULTING IN BLINDNESS OF ONE EYE.

EDWARD B. HECKEL, M.D.

PITTSBURGH, PA.

Attention was directed to this subject, by seeing during the last few years, a number of cases (ten in all), in which the principal points in the histories were practically identical; the chief thing complained of was exactly the same, namely blindness in one eye, following a slight head injury, so that we may safely group them all together and consider them as one case.

In all of them there was a history of trauma—a bump over the temporal region, or over the eyebrow, or a knock on the jaw, in some sufficiently severe to stun the patient, some three to six months previous to seeing him, but no external evidence of injury at the time of examination. On inquiry, we always learned that there had been little or no external evidence of any injury at any time. The universal complaint, however, was that there was little or no vision in one eye immediately after the injury.

The examination of these cases showed a striking similarity; the pupils were equal, they reacted to light, accommodation and convergence; the consensual pupillary reflex, however, was always absent in the good eyes. Ophthalmoscopic examination showed clear media and negative fundi in the good eyes, but atrophy of the optic disc in the blind eyes. The good eyes remained normal for vision, color sense and fields. The eye movements were normal. X-ray pictures were made in all of them with negative results.

From an anatomic standpoint, the only lesion which could result in blindness of one eye, as detailed above, must lie in front of the chiasm, at or near the apex of the orbit, and most likely the result of a fracture, yet we had no proof for our hypothesis, the supposition being that all of these cases were seen and X-rayed too long a time after the injury to show any fracture.

We had the privilege, however, of seeing and observing the case of a young man who had been struck by a railroad train at a grade crossing; he was stunned but was not unconscious. When we saw him, just two weeks after the accident, there was still some evidence of a slight subdermal hemorrhage of the lids of each eye. The eyes were negative in appearance, except a slight subconjunctival hemorrhage of the left eye. The pupils were equal and reacted to light, accommodation and to convergence. The right pupil, however, failed to react consensually, while the left did. The left eyeball deviated very slightly down and out. Ophthalmoscopic examination showed the media clear and the fundi negative (the left optic disc has since then become atrophic, and the eye

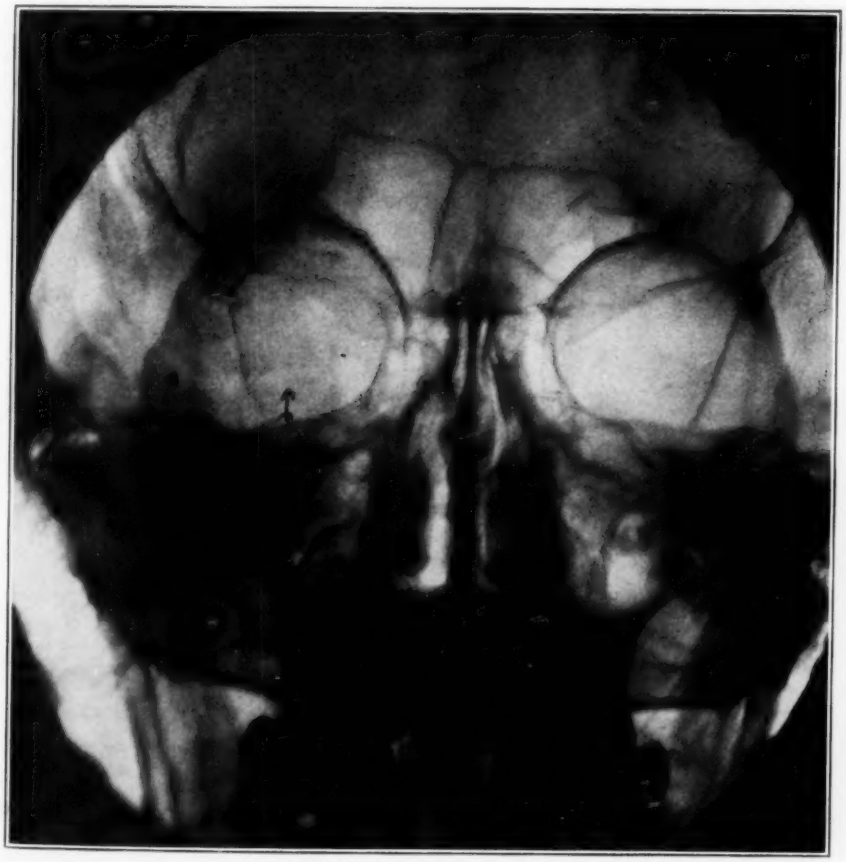

Fig. 1.

movements normal). R. V. = 20/20 —, with normal color perception and normal fields. L. V. = 0.

The X-ray in this case (Fig. I and Fig. II) shows a very extensive comminuted fracture of the left orbit. The pictures show three distinct lines of fracture, all of these extending from the marginal line of the orbit to the optic foramen. Two of the lines of fracture are in the external wall, and one in the floor of the orbit. The apex of the orbit is crushed inward and encroaches upon the external margin of the optic foramen, as designated in the illustrations by means of arrows. X-ray evi-

Fig. 2.

dence of these fractures has since disappeared; the optic atrophy and blindness of the eye remains.

This last case illustrates beautifully the great advantage of seeing and X-raying these cases early, and establishes a causative factor in the other cases even with negative X-ray findings, for it is our firm belief that had we had the opportunity of seeing the other cases early, the X-ray would have demonstrated what it did in the case just recorded.

DISCUSSION.

Dr. Wm. L. Benedict, Rochester, Minn.: Lesions of the middle fossa of the cranial cavity may be brought about by contusion, concussion, or pressure exerted from internal sources, such as a rapidly growing tumor. It is almost impossible to demonstrate by roentgenograms definite fractures as the result of compression; it is also impossible to demonstrate, with any degree of accuracy, lesions of the cranial cavity in case of malignancy such as rapidly growing tumors. As you know, a fracture of the internal table of the skull may occur without fracture of the external table, caused by a blow on the head by a blunt object such as a ball bat, or following an automobile accident or a skating accident, a fall in which the head strikes a solid object not too forcibly. It is almost impossible to demonstrate these fractures of the internal table by roentgenograms, except rather soon after the accident. There are many cases of monocular blindness of uncertain etiology, which can probably be traced to apparently minor injuries. It is not always, however, that such injuries as have been reported by Dr. Heckel will result in blindness of one eye without evidence of change about the optic disc. Only recently I had the privilege of seeing an eleven year old girl who had been struck above the temple by the seat of a swing, at one of the public playgrounds. A week following the injury, she had edema of the optic nerve on the side on which she was struck, which later was followed by atrophy. If ophthalmoscopic examination shows a simple atrophy without evidence of inflammation, then the inference which Dr. Heckel brings is correct, that the lesion must occur near the apex of the orbit. Unfortunately, the photograph he showed did not show any change in the optic foramen. Whether this injury is due actually to pressure near the apex of the orbit, whether it is brought about by an edema, or whether by hemorrhage into the vaginal sheath, is questionable. We do know, that following some operative procedure in the middle fossa, the vaginal sheath of the optic nerve will be filled with hemorrhage. This has been noted particularly after operation for tumors in the chiasmal region.

Not all cases of monocular blindness, as described by Dr. Heckel, can be attributed to injury, unless there is very good evidence of blindness following immediately afterwards. You will recall some cases of ring tumor reported a year ago by Dr. Dandy, that caused compression of the optic nerve in the region of the optic foramen. In cases which have been blind for some time, the importance of injury may be overemphasized.

DR. J. S. LICHTENBERG, Kansas City, Mo.: It is a well known fact in ophthalmic literature, that apparently trivial injuries of the orbit cause fractures which extend into the optic foramen and cause atrophy. I want to call attention to an article by Van der Hoeve, in which he sets forth an improved method of Roentgen technic in this region of the optic foramen.

DR. HARRY S. GRADLE, Chicago: I wish to take issue with two points that have been brought out in the paper and discussion. In the first place, some of the roentgenographic evidence of fracture of the apex of the orbit depend to a great extent upon the careful technic of the roentgenologist. Within the first six weeks after the injury, as a rule, the fracture can be manifest. Later on, months and eventually years, the determination of fracture of the apex of the orbit depends upon the presence of callus formation. Personally, I have had a case as long as five or six years after the fracture, where careful stereoscopic roentgenograms showed a line of fracture very clearly. In the early stages, within the first two or three weeks, of fracture of the apex, a study of the case, with a careful investigation of all of the factors concerned with the eye, will as a rule allow of a beautiful diagnosis and prognosis. We can tell whether the vision is reduced by the edema of the nerve, or whether it is reduced by hemorrhage at the apex, and as a result we are able to give a prognosis as to the ultimate vision. We can tell the location of that fracture, to a certain extent, from muscular anomalies that may be present. Furthermore, we can also tell, from the extent of the muscular anomalies, approximately how much vision will return. This, combined with the roentgenographic evidence, will allow us to give a rather accurate prognosis within the first three to five weeks after the fracture of the apex of the orbit.

I believe these fractures are much more common than we have recognized in the past, and I believe, if we will cooperate with the roentgenologist in careful stereoscopic examinations, especially from the lateral aspect, we can show the presence of fracture and can make our prognosis for the benefit of the patient earlier than in the past.

CATARACTS: OPERATION FOR CONGENITAL AND JUVENILE.

F. W. Dean, B.S., M.D., F.A.C.S.

COUNCIL BLUFFS, IOWA.

It is not my purpose to discuss the etiology of juvenile and congenital cataracts, nor to consider the several types of cataracts which are found in the young. I take up the subject at the point where the vision in a young person is so poor, because of a cataract, that it is thought advisable to remove the lens in order to secure better vision.

The removal of the lens is accomplished either by a linear extraction, by discission and absorption, or occasionally by suction. A linear extraction of a lens from the eye of a child whose postoperative behavior cannot be controlled, should not be undertaken lightly. If such an operation is done with an iridectomy, there is a cosmetic defect which must be carried thru life. On account of the lack of cooperation of the child, if a simple extraction is done, a prolapsed iris and other accidental bad results may follow. The method most commonly employed is discission, allowing the lens to be dissolved and absorbed by the aqueous. This procedure is far from an ideal one. Except in the very young, the discission must be repeated a number of times. The length of time required for absorption varies according to the extent that the capsule has been opened and the lens tissue broken up, and to the age of the patient. When using this method of operating, it is not safe to make a large opening in the capsule and to stir the lens tissue to any great extent, for fear of a swelling of the lens and a consequent glaucoma. When a slight opening is made in the capsule, with little or no breaking up of the lens substance, a long time is required for the absorption, and repeated needlings must be done. The younger the patient, the shorter the time required for the complete absorption of the lens.

Dr. F. A. Callan says, (System of Ophthalmic Operations, Wood, Vol. II, p. 1294), "The question of time with children

is of little importance, so that there is no excuse for hastening the process by numerous or extensive needlings." I cannot agree with Dr. Callan that the question of time with children is of little importance.

Industrially, he may be right, but because of the long time required for absorption to take place, and the more or less constant irritation and inflammation of the eye during this long period, there follows a thickened capsule which must be removed to secure good vision. This constant irritation brings about a thickening of the capsule, in the same manner that anterior polar cataracts are formed by the contact of the lens capsule with the cornea plus an irritation in prenatal life. The removal of this thickened capsule is another serious problem. It may contain calcareous deposits, and must be removed and not simply opened. The removal of a tough, leathery capsule, with the necessity of loosening its attachments from the vicinity of the ciliary body, is not without its hazards.

Dr. Ziegler, (Transactions of the Ophthalmological Society of the United Kingdom, Vol. 45, p. 154), advocates a large V-shaped opening in the lens, the cuts being made completely thru the lens, including both the anterior and posterior capsules. He asserts that with such an opening the absorption is rapid, that the tension of the eye is lowered rather than raised by the swelling of the lens, that an aftercataract does not occur, and that absorption is complete in one month. I have had no experience with this operation.

While in London, in 1893, I picked up an instrument brought out in 1863 by R. L. Teale. This instrument consists of a glass canula, on one end of which is attached a silver tip which is hollow and has an opening at one side of the distal end. At the other end of the glass canula is a rubber tube, into which is inserted a mouth piece. The suction method of removing the lens with this instrument or other models, was used by Teale, Bowman, Lawson and others in England. It was also used to some extent in Belgium and France. Dr. Hasket Derby (Handbook of the Medical Science, 1885) reported 107 cases of cataracts extracted by suction at the Massachusetts Eye and Ear Infirmary.

For a number of years, I have used the suction method with the Teale type instrument, for the removal of soft cataracts, and much prefer its use to the discission and watchful

waiting method. The instrument I bought in London has a metal tip cemented on a glass barrel and is hard to clean. V. Mueller, of Chicago, has made for me an instrument with a silver barrel, which can be easily cleaned and boiled. No change was made in the instrument except to aid in its sterilization. (I would not use a suction instrument with a piston from fear that the piston might stick and, freeing itself suddenly, draw out more than the lens tissue.) The action of the Teale instrument is at all times under perfect control. My technic is as follows: First atropin is used, then the eye is desensitized with a local anesthetic, or a general anesthetic is used, as the case requires. With a needle knife, the cornea is punctured about 2 mm. from the limbus in the upper temporal segment. I believe there is no virtue in entering thru the con-

junctiva and the anterior chamber at the angle. In such a puncture, the iris may follow the blade out, leaving a prolapse under the conjunctiva disfiguring the eye, if no other trouble results. Furthermore, it is impossible to stir up more than the anterior part of the lens, unless the puncture is made more anteriorly. After the anterior chamber is entered thru the cornea, the point of the needle knife is passed thru the pupil, the capsule is freely opened, and the lens tissue thoroly incised and exposed to the action of the aqueous. The needle knife must not cut thru the lens into the vitreous. The atropin is continued. After waiting a few days, the lens will be found swollen, softened, and broken up by the aqueous. The second step of the operation is then undertaken.

Following another anesthesia, an oblique incision is made thru the cornea, in the location of the former puncture, with a Graefe knife. When operating an anterior polar cataract, one is able to free the cataract from the capsule at the time the corneal opening is made if a Graefe knife is used, a procedure well nigh impossible with a keratome. The incision is made only large enough to admit the point of the suction instrument. The glass mouth piece is held between the lips by the teeth. The

tip is passed thru the opening in the cornea, and the opening in the tip is brought under and in contact with the lens substance to be removed. A slight movement of the tongue draws the lens fragment into the barrel. The tip is moved from one particle of lens to another until all has been removed. This is done with one introduction of the tip into the anterior chamber. Care should be taken to keep the opening of the tip forward toward the cornea and so enable the operator to see clearly what is drawn into it. I use a binocular loupe to better see the progress. If one is fearful of an infection following a corneal incision, the lips of the wound can be cauterized and covered with conjunctiva. I have not thought that procedure necessary. Atropin is continued until the eye is quiet. If later there develops an aftercataract, which has occurred in about 50 per cent of my cases, it will be found thin and easily opened. The question presents itself as to whether congenital cataracts might not produce an anaphylaxis as do some senile cataracts. I have never seen such a reaction in any of my cases. Following the removal of the congenital and juvenile cataracts with the Teale instrument and the technic described, I have been so fortunate as to have secured good results. That is, there have been no accidents during operation, and no infection or inflammation following the operation. There have been no cosmetic defects, and in every case useful vision has been secured. One possible exception to this statement is case number five, reported later in this paper, in which vision has not been checked. To be sure, many of the eyes have high degrees of astigmatism and are otherwise defective, and yet these patients secure the benefit of clear media. As a consequence of these results, I regard the discission, followed in a few days by the withdrawal of the lens with the Teale type instrument, the proper operation for congenital and juvenile cataracts. From my cases I have selected seven patients, and will report in detail the progress of the ten eyes operated upon, as they show the results of the operation in varying types and conditions.

Case 1. Sept. 8th, 1914. D. S., boy, 10 yrs. Congenital anterior polar cataract in each eye. Vision too defective for progress in school. Same date, Sept. 8th, 1914, needled lens of left eye. Sept. 11th, three days later, sucked out lens and also the thickened capsular cataract. Oct. 2nd, 1914, needled lens of the right eye. Oct. 5th, three days later, drew out lens

and capsular cataract. Nov. 3rd, 1914, gave bifocal lenses for each eye, R. and L. + 12.00/15.00 ⌒ + 3.00 ax. 90. Vision of each eye with this correction equaled 20/40 and J.2. April 21st, 1925, ten years after operation, opened very thin aftercataract in right eye. Left eye was clear. The boy has finished his high school course. Was last seen Dec. 5th, 1925. At that time, his vision with same correction as given in November, 1914, was: Right 20/30 and J.1; left 20/20, 2 letters, and J.1.

Case II. April 11th, 1917. G. G., boy, 11 yrs. Had a haziness of each lens. Had had a lightning stroke a few months previous. Feb. 20th, 1918, cataracts in each eye had reduced vision to less than 20/200. Owing to home conditions, the boy was sent to hospital and both lenses were needled under a general anesthetic. Three days later, Feb. 23rd, lens tissue was sucked out, using a general anesthetic. May 27th, 1918, gave lenses for distance, Rt. + 12.00 ⌒ + 2.00 ax. 90; left, + 12.00 ⌒ + 1.50 cyl. axis 90. Patient did not return for reading glasses until brought in by truant officer, March 13th, 1920, two years after his operation. Adding + 3.00 to his distance, correction in bifocals, gave him in each eye 20/20 and J.1.

Case III. March 13th, 1918. Miss N. C., 19 yrs. Patient was in hospital with acute diabetes. Had cataracts in each eye, which had developed rapidly during her illness. Right eye, vision equaled objects. In the left eye, the lens appeared as a mature cataract. I needled the left lens under a local anesthetic. April 7th, the lens was sucked out. April 29th, fitted eye with bifocal lens, + 13.00/16.00, vision equals 20/30 and J. 1. Patient moved to another state and operation was not done on the right eye.

Case IV. March 16th, 1918. C. T., boy, 10 yrs. The right lens had been needled by a well known ophthalmologist years previous to my examination. The eye had been entered under the conjunctiva and at the angle of the anterior chamber. The iris was prolapsed under the conjunctiva and the pupil elongated outward. There was a thick leathery aftercataract shutting off vision. I needled the lens of the left eye. March 23rd, I found that I had not incised the lens thoroly enough, so repeated the needling. April 5th, 1918 sucked out the broken lens. April 30th, fitted the eye with bifocal lens with correction, + 13.00/16.00 ⌒ + 4.00 ax. 90; his vision equaled 20/30 and J. 1.

CASE V. March 7th, 1925. Mr. R. S., 36 yrs. Had blow on right eye from chip of wood while chopping. Capsule of the lens was ruptured. There was a beginning cataract and some swelling of lens. Patient insisted on returning to his home, twenty-five miles distant. He was given a tube of atropin ointment and told to return in two days. He returned April 1st, nearly four weeks after the injury. He complained of intense pain, which began three days before. I found the anterior chamber filled with softened lens substance and an intraocular pressure of 130, McLean tonometer. I did a paracentesis of the cornea and reduced the tension by letting out the aqueous, a drop at a time. After the tension was reduced, I enlarged the paracentesis opening and sucked out all of the softened lens. He was sent to the hospital for observation. He had no pain or increased tension after the lens was removed; the eye was quiet and there was no cosmetic defect. He left the hospital on the second day. I have not had an opportunity to check up his vision.

CASE VI. April 11th, 1925. Miss F. E., age 25 yrs. Had nuclear cataracts in each eye. Had never seen well. Vision, R. and L. equals 20/70. April 15th, 1925, needled right lens. April 22nd, seven days after needling, sucked out the lens. May 20th, needled lens of left eye. May 23rd, repeated needling left lens. May 25th, five days after the first needling, sucked out the lens. June 22nd, bifocals were prescribed. R. + 11.50/14.50; L. + 10.00/13.00 ⌣ + 1.50 axis 15. July 28th opened thin aftercataract in left eye. Vision with her correction was 20/20 and J.1 in each eye.

CASE VII. Aug. 21st, 1924. F. J., boy, age 14 yrs. Struck iron garden swing with hammer. A particle of steel penetrated cornea and iris of left eye about 2 mm. from the inner limbus, and was located by the X-ray near the center of the vitreous body. With a Lancaster hand magnet, the particle of steel was removed thru a slightly enlarged entrance opening. The steel had evidently passed thru the lens, as a cataract developed. Jan. 21st, 1926, seventeen months after the injury, I needled the lens with the object of relieving the patient of a white pupil and putting the eye in a condition for possible future usefulness. Jan. 25th, four days after the needling, I sucked out the lens substance. Patient was away from school one week only. March 27th, there was a very thin aftercataract which was perhaps no more than a wrinkling

of the posterior capsule. This was not opened, as patient will use other eye. Vision with + 10.00/13.00 ⌒ + 1.50 ax. 110 was 20/23 and J.2 in spite of the thin aftercataract.

My conclusions are: That the suction method of operation is suitable to any form of juvenile or congenital cataract; that it is not necessary to keep the patient, at any age up to twenty-five years, under operative treatment longer than seven days, if the lens is thoroly incised; that it is possible to suck out the lens of a man thirty-six years old at one sitting; and that the results of the suction operation for congenital and juvenile cataracts are wholly satisfactory.

DISCUSSION.

Dr James M. Patton, Omaha: Dr. Dean has mentioned the various types of procedure with which we are all familiar in dealing with congenital cataract. It is interesting to note, that these various procedures, in the hands of those who are acquainted with them, almost always give satisfactory results. The personal equation enters into it very largely. I can not claim the experience or the skill that Dr. Dean has shown in the management of this technic. However, I have used it enough to convince myself that it has real merit, and that in this type of case, congenital cataracts which we have softened, or soft traumatic cataracts, we have a method which is of value. The opening in the anterior chamber is small, the field of operation is constantly under our view, there is no need for haste, and we can proceed cautiously and completely to the removal of the opaque substance. The opening in the instrument is presented forward toward the cornea and there is no need to disturb the vitreous if one is at all careful.

As to the question of protecting the postoperative wound with one of the various types of conjunctival flaps, I would not take issue with the author on that, but personally I do like some form of conjunctival protection for these wounds. Dr. Dean's excellent results, however, show that this protection is not at all essential; but here, again, the question of technic can be varied to suit the ideas of the individual operator.

There is one situation in which I think this instrument is of particular advantage, and that is in the case of the soft cataract, secondary to trauma, where you suspect a rupture of the zonule. We all know that in these cases, if we resort to the usual pressure method of expression, there is possibility of escape of vitreous before we have thoroly relieved the anterior chamber and the lens capsule of its contents. Here we have a small opening, a clear field, and plenty of time for delicate manipulation, so that we can clear out the lens substance more completely and with less danger of vitreous loss than where we resort to the ordinary pressure method.

There is another circumstance in which this method is of value, viz., where we have an infected lens in young people, where we know the lens substance is soft. We all know how unsatisfactory it is to try to express an infected lens under those circumstances. I have on occasions resorted

to the suction method for this purpose, and find that I can suck out the infected material so that the natural resistance of the patient may overcome the infective condition.

Just a word about the instrument itself. As with all instruments, sometimes the one supplied by the dealer is not exactly the same as the author describes. The instrument makers are not always able to build them accurately, and sometimes they sacrifice the lumen of the instrument to lightness and delicacy. You must be sure that the instrument has a sufficient lumen, so that it will not be easily blocked by lens material or capsule.

The procedure has definite merit, and I wish to express my personal appreciation to Dr. Dean for bringing it to our attention.

Dr. Wm. H. Crisp, Denver: One very brief suggestion as to the apparatus employed. The rubber tube shown is rather short, and there might at times be danger of saliva running down the tube into the eye which is being operated upon. If the rubber tube were lengthened so as to allow it to form a dependent U, this danger would be obviated.

Dr. Walter B. Lancaster, Boston: I have had experience with both this method and with Dr. Ziegler's thru-and-thru incision. As you could judge from the quotations made by the author, suction was used in Boston a good deal at one time, Dr. Haskett Derby reporting a large number of cases in 1885. When I began practice there, it was in general use, and I remember devising a little modification of the tip so that it could be boiled. I made it entirely of glass, but a little shorter. I have forgotten now why I gave it up, but the fact is that they have not used it much in Boston for a good many years. I had a case within a couple of years where I was strongly tempted to use it in senile cataract, because there was considerable cortical substance left; but the same argument applied there as in other cases—if you give Nature a chance, this material will be absorbed, and this policy is a little safer in the average hands.

As to the Ziegler method, I have tried it, but I do not think I shall try it again. The case turned out well, but it gave me a good deal of anxiety, and I cannot see that it is wise to cut thru into the vitreous, when if you do an ordinary discission, your eye is protected by the posterior capsule, so the vitreous need not enter the anterior chamber, at least until the time comes for the final discission.

Dr. Wm. L. Benedict, Rochester, Minn.: The remarks of Dr. Lancaster prompt me to say a few words in regard to the Ziegler operation, which I have been doing for several years. I never have seen but one bad result from the thru-and-thru incision, in which we attempt, as Dr. Ziegler has suggested, to go thru the entire lens. I fail to see any difference whether we go thru the posterior capsule at the first incision, or whether we go thru it some time later. I believe I would rather have one operation than make two attempts, and I see no contraindication. The only failure I have had was in a child who was marasmic, in which I operated both eyes, one of which had a perfect result and the other one went on to phthisis.

I can hardly see the value of making an incision thru the cornea. With Dr. Ziegler's technic of making an incision at the limbus, I have never seen a prolapse of the iris. The operation, in most instances, can be

done without loss of the anterior chamber, and without any more risk of infection than from a corneal incision, and we avoid the tendency to vascularity which sometimes occurs with a corneal puncture 2 millimeters from the limbus.

Lens material can be evacuated by the suction method, but I prefer irrigation. I feel that by irrigating the chamber with a specially devised irrigator, there is no danger of increasing the intraocular pressure, there is no danger of making the intraocular pressure too low, the iris can be kept away fom the posterior wall of the cornea, and the material that has coagulated is easy of access, and by the use of a silver spatula sufficient room can be made for complete evacuation of the contents. This can be done by irrigation without pressure and without changing materially the normal tension of the eye.

Dr. J. S. Lichtenberg, Kansas City, Mo.: Just one point that was brought out on the subject of sensitization to lens protein. This subject has been worked out by Verhoeff and Lemoine, and the material for the test is now being manufactured by well known commercial houses and can be obtained on the market. It is very simple to tell whether a patient is sensitive to lens protein or not.

Dr. Wm. H. Wilder, Chicago: I think it is very appropriate that this old method of suction for the removal of soft cataracts should be brought to our attention again. The method should also be applicable for the removal of lens substance from the anterior chamber in traumatic cataract following penetrating wounds of the cornea. In such cases, particularly if the eye is lens sensitive, there is considerable reaction, and healing is delayed because of the presence of lens substance in the anterior chamber. In such cases, in addition to the iridocyclitis that is excited or aggravated by the presence of the lens substance, there is danger of increased tension that would tend to impede the absorption of the lens material. The favorable results that follow show that it is good surgery to remove this offending material, and I think this method of suction might often be used thru a small wound instead of the method of expression and irrigation that have proven useful in such cases.

I think the suggestion made as to increasing the length of the tube is a very pertinent one. The drop in the tube would act as a trap in a waste pipe to prevent any back flow.

As to the "thru-and-thru" incision of Ziegler in the treatment of congenital cataract, I agree with Dr. Lancaster's conclusions. It has seemed to me, that when the vitreous is allowed to mix with the lens substance, there was marked delay in the absorption of the lens. For this reason, I have usually refrained from a final discission that would connect the vitreous with the anterior chamber until most or all of the cortical substance has been absorbed.

Dr. Walter B. Lancaster, Boston: The condition that gave me anxiety in the case I referred to, which was the last one I did, was an acute rise of tension. The glaucoma necessitated some interference. I think it is quite obvious that evacuating the lens material when there exists a good sized opening into the vitreous is not so advantageous to the patient as when you have the posterior capsule intact. If you irrigate, you

are likely to wash out vitreous as well as cortical substance. They come out equally well—in fact, the vitreous comes out a little the easier.

Dr. F. W. Dean, Council Bluffs, Ia. (closing): I am very thankful for the discussion and for the points brought out. Dr. Benedict speaks of doing the Ziegler operation, saying it is no more dangerous to cut into the vitreous the first time than it is when you cut into it for an after-cataract.

In the Ziegler operation, the vitreous is mixed with the lens substance. If the lens must be removed, it cannot well be washed out if there is a large opening into the vitreous. In spite of the fact that Dr. Ziegler said the tension of the eye is never raised, I cannot believe it.

It is not necessary to open an aftercataract in every case. When it is done, the danger of an infection is due to some of the vitreous getting into the corneal wound. That is one reason why I am careful not to cut into the vitreous when doing a discission.

The point about the length of the rubber tube used with the instrument is good. I think, however, the length of the tube may be left to the judgment of the individual operator.

MELANOTIC SARCOMA OF THE CHOROID. REPORT OF TWO CASES. BIBLIOGRAPHY.

C. NORMAN HOWARD, M.D.

WARSAW, INDIANA.

It would seem that the first case of melanotic sarcoma of the choroid was reported by Gunther in 1824. A careful review of literature discloses that from then until January 1 of this year, (1926), there has been at least 407 articles written on this subject.

While melanotic sarcoma is regarded as the most frequent tumor of the choroid, still its actual occurrence is rare. B. Castresana, in an article appearing in Siglo Medico, Madrid, in 1916, states that he has encountered five or six to each ten thousand eye patients, or about 1/20 of one per cent. Again, Dr. John F. Gipner, pathologist at the Mayo Clinic, writes me that only 16 cases of melanotic sarcoma of the choroid were encountered among 152,651 patients seen in their eye department from 1920 to 1925 inclusive. Apparently the frequency of occurrence there is about 1/100 of one per cent. These figures, however, include a large number of medical cases seen only for fundus examination. The primary eye cases alone were 39,854, which would increase the percentage to about 1/25 of one per cent, or 4 out of every 10,000 primary eye cases seen at the Mayo's.

It is interesting to note that Dr. Gipner, in giving me these figures, did not use the term, melanotic sarcoma of the choroid, but melanotic epithelioma, instead.

It will be remembered that, in 1924, Dr. Mary S. Knight presented an able treatise on melanotic neoplasms of the eye before the Section of Ophthalmology of the American Medical Association. Dr. Knight's findings were based on work done at the Mayo Clinic. Her contention was, that altho commonly called melanotic *sarcoma*, nevertheless these pigmented tumors should be more properly termed melanotic *epitheliomas*. Dr. Knight said, "Wherever studied, melanin has been produced by ectodermal cells. * * * Therefore,

tumors capable of producing pigment arising in the eye should be called melanotic epitheliomas." I mention this, not only for its own interest and value, but also to dispel any thought that melanotic epithelioma of the choroid is different from melanotic sarcoma of the choroid. As I understand it, they are the same, but we are witnessing an effort to present a more accurate name for these pigmented malignant growths of the choroid, commonly called sarcomas.

There seems to be a general agreement that local recurrences in the orbit are comparatively rare, following enucleation for melanotic sarcoma of the choroid. There is, however, a difference of opinion as to the frequency of metastasis.

De Schweinitz, in his text book, page 406, says: "Prognosis: Removal of an eye for choroidal sarcoma results in a cure in from 25 to 30 per cent of the cases, altho statistics on this point vary greatly. Hirschberg's published statistics may be briefly summarized as follows: Local recurrence, 2.5 per cent; metastasis 41.5 per cent; cure 56 per cent."

Von Hippel is quoted on page 340 of the 1924 Ophthalmic Year Book, as being of the opinion that most reported cases are under observation too short a time to prove that metastasis has not occurred. In one of his cases, it appeared eighteen years after enucleation.

There has been some thought that the spindle cell melanotic sarcoma of the choroid is not apt to produce metastasis so soon as the mixed cell type. This idea happens to be borne out in the two cases I am reporting.

Report of Cases.

Case 1 is a mixed cell melanotic sarcoma of the choroid followed by metastasis.

The patient was a man aged 57. He gave a history of having first noticed a visual disturbance in the left eye one and a half years before I first saw him on June 18, 1924. I found the vision in this left eye to be 13/200. It was not materially improved by lenses. There was a mass in the upper temporal region pushing the retina before it toward the median line. On June 30, 1924, I removed this left eye along with as much of the optic nerve as possible—about 5 mm. of it. On opening the eyeball, there was revealed a growth of cauliflower appearance, about 9x10x12 mm. in size. It was adherent to the choroid. The external surface of the sclera showed no sign of extension to it.

The eyeball was sent to the Army Medical Museum. Under date of August 21, 1924, the following report on the growth from Dr. F. H. Verhoeff of Boston was transmitted to me by the Army Medical Museum:

"Pathologic diagnosis: Melanotic mixed cell sarcoma of choroid. The tumor shows numerous small areas of necrosis. Behind the tumor, the sclera shows perivascular invasion by the tumor. The cross section of the optic nerve shows no invasion of the nerve by the tumor."

On January 17, 1925, the Museum transmitted an additional report on this growth from Dr. Verhoeff:

"Pathologic Report: Second section: This is a celloidin

Fig. 1. (Case 1). Microphotograph. Melanotic mixed cell sarcoma of choroid. 75 mm. lens.

section thru the entire globe. In this section, the tumor cells are practically all large round cells, identical in appearance with those of a melanotic malignant nevus of the skin. The tumor shows large areas of necrosis. The tumor has not invaded the sclera to any appreciable extent. It extends to within about 3 mm. of the optic disc, but has not invaded the optic disc or nerve. The optic disc is not cupped." The Army Medical Museum has this specimen on file as its Accession No. 21880.

Thru the patient's home physician, Dr. Norman L. Reynolds of Warsaw, Ind., I learned that there was never any return locally in the orbit, but that the patient died from metastasis. It seems that the metastasis first began to make itself definitely manifest about four months after the enucleation, thru severe pain on the left side, in the region of about the fourth or fifth dorsal vertebra. Three weeks before his

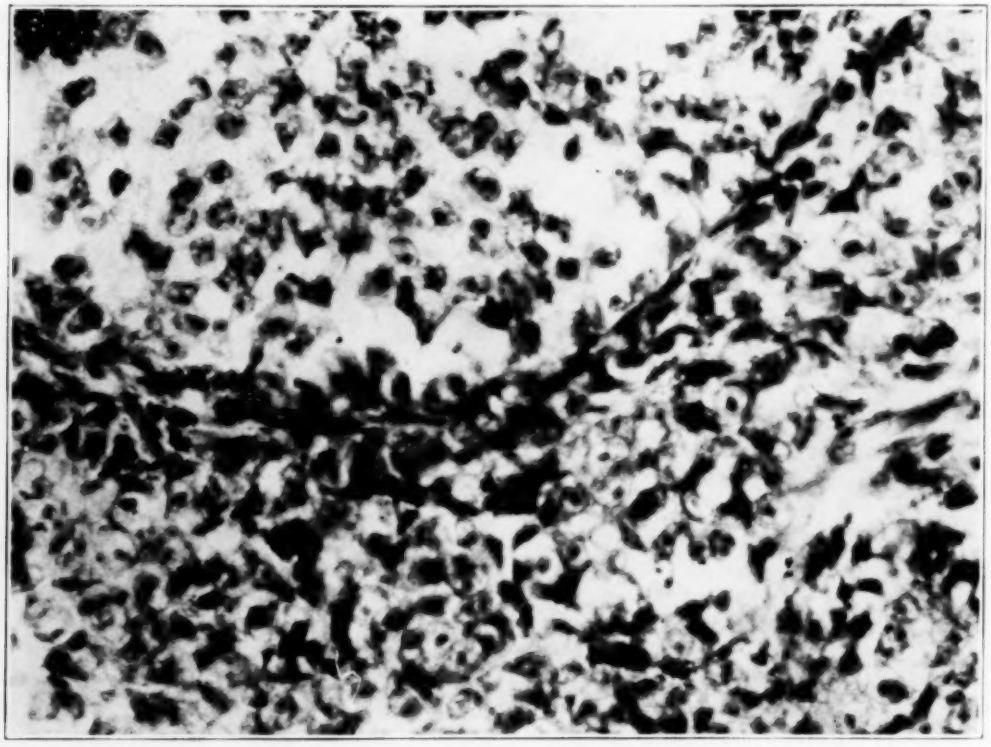

Fig. 2. (Case 1). Microphotograph. Melanotic mixed cell sarcoma of choroid. Lens 8 mm. Ocular 6. Bellows 78.

death, there was some difficulty in moving the left foot. Several days after that, total paraplegia developed suddenly, so that there was complete loss of motion and sensation from the waist down, with no control of rectum and bladder, the metastasis apparently involving the spinal cord. The above symptoms persisted until the patient's death on April 11, 1925, being less than two and a half years from the time he first noticed a slight disturbance in his left eye, and a little less than ten months after the enucleation.

The microscopic findings of the growth are shown in Figures 1, 2 and 3, prepared thru the courtesy of the Army Medical Museum. They show the mixed cell type of melanotic sarcoma of the choroid.

CASE 2 is a spindle cell melanotic sarcoma of the choroid with metastasis delayed or escaped.

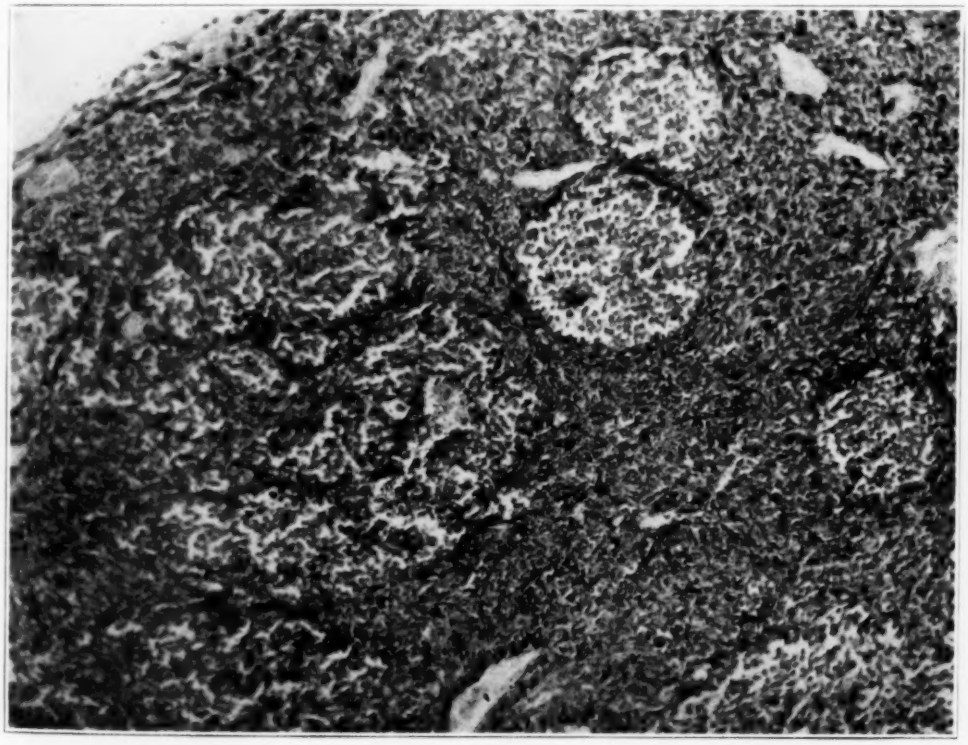

Fig. 3. (Case 1). Microphotograph. Melanotic mixed cell sarcoma of choroid. Lens A. A. Ocular 6. Bellows 78.

On October 3, 1922, the patient, aged 63, came to the office complaining of poor vision in the right eye. She gave a history of having severely bumped the eye three weeks previously. The vision in that right eye was found to be 6/200. The eye was also found to have a partial detachment of the retina. Whether the injury simply called the patient's attention to the poor vision, or whether it was an etiologic factor, I do not know. The eye became gradually worse, with the vision decreasing, and finally glaucoma developed. I enucleated the eye on March 25, 1923, a little over six months after the patient first noticed trouble.

On opening the eye ball after removal from the orbit, a pigmented tumor was found adherent to the choroid. It was attached by a broad pedicle and measured 9x10x5 mm. The mass was entirely within the eyeball. That is, there was no growth seen on the outside of the eyeball, nor was there any mass felt in the orbit.

The eyeball was sent to the Army Medical Museum. After a preliminary report, I received the following letter on May 1, 1923:

"Dear Doctor Howard:

"In my opinion, the case of Mrs. A. W. of Atwood, Ind., [Case 2] presents histologic and merchanical factors which

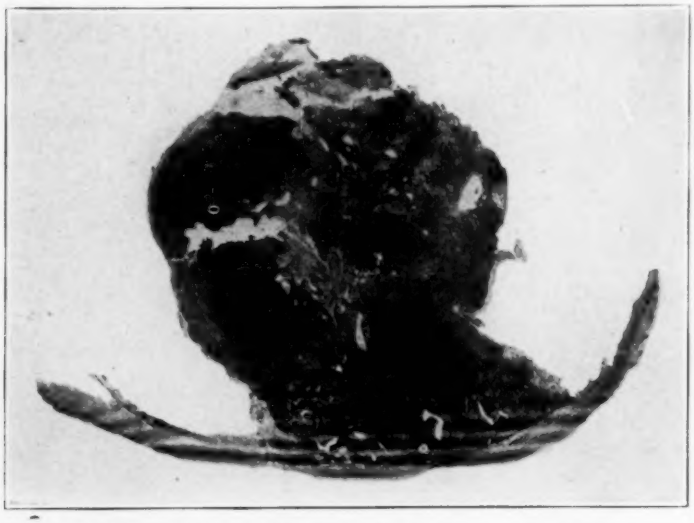

Fig. 4. (Case 2). Microphotograph. Melanotic spindle cell sarcoma of choroid. 75 mm. lens.

would indicate that there had been no metastases of this tumor. The spindle cell type is comparatively slow to metastasize except along lymphatics, and in this case had not penetrated the connective tissue envelope of the eye. A careful search along the optic nerve failed to reveal any evidences of metastasis. It would be my hope, from the early recognition of this tumor and its small and circumscribed character, that this patient will never hear from this condition again.

"The whole of the material in this case will be sent to Dr. F. H. Verhoeff for consultation and special request will be made upon him for further information along these lines.

Sincerely yours,

James F. Coupal,
Major, Medical Corps, U. S. A.
Curator, Army Medical Museum."

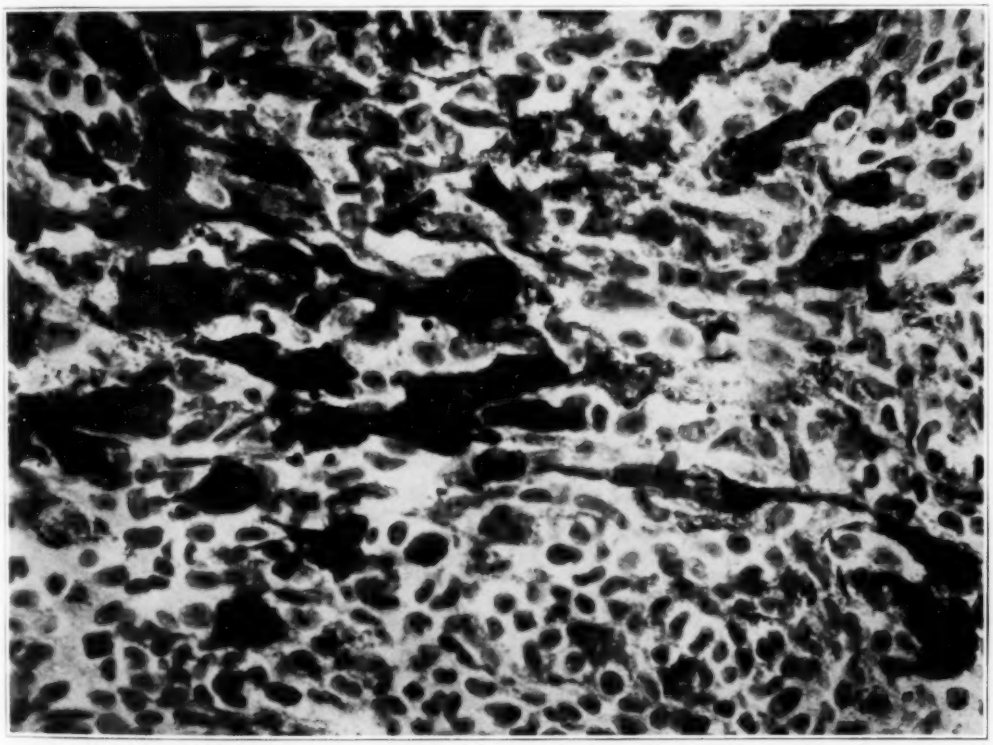

Fig. 5. (Case 2). Microphotograph. Melanotic spindle cell sarcoma of choroid. Area in which cells are producing melanin. Lens 8. Ocular 6. Bellows 78.

The report from Dr. F. H. Verhoeff was transmitted thru the Army Medical Museum under date of May 28, 1923. Dr. Verhoeff stated:

"The tumor is a melanotic spindle cell sarcoma of the choroid presenting no especially unusual features. It shows large areas of necrosis, contains numerous large blood spaces lined by endothelium, to which the tumor cells are closely applied. Into some of these blood spaces, the tumor cells have apparently proliferated. The tumor has invaded the sclera to a very slight extent.

The anterior part of the eye shows early peripheral anterior synechia, which has been freed in the preparation of the specimen. No regional metastases are found in the anterior part of the eye. The optic nerve shows no invasion by the tumor.

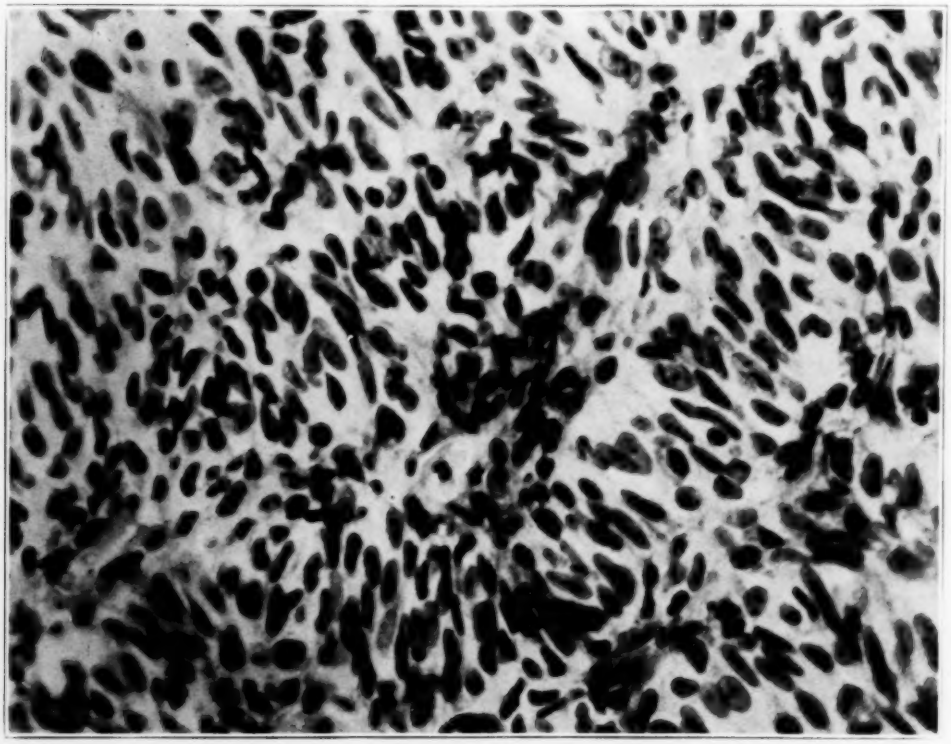

Fig. 6. (Case 2). Microphotograph. Melanotic spindle cell sarcoma of choroid. Area in which cells are *not* producing melanin. Lens 8 mm. Ocular 6. Bellows 78.

Pathologic Diagnosis: Melanotic spindle cell sarcoma of choroid. Early secondary glaucoma.

Prognosis: Relatively favorable, but the possibility that general metastases have occurred cannot be absolutely excluded."

Dr. T. J. Clutter, of Mentone, Ind., the patient's home physician, tells me that she is well, having apparently no local recurrence in the orbit or general symptoms. It is now over three and a half years since the enucleation, and four years after she first noticed trouble with the eye.

Figures 4, 5, 6 and 7, obtained from this growth were also prepared by the Army Medical Museum, the specimen being Accession No. 19927. These figures serve to illustrate the spindle cell type of melanotic sarcoma of the choroid.

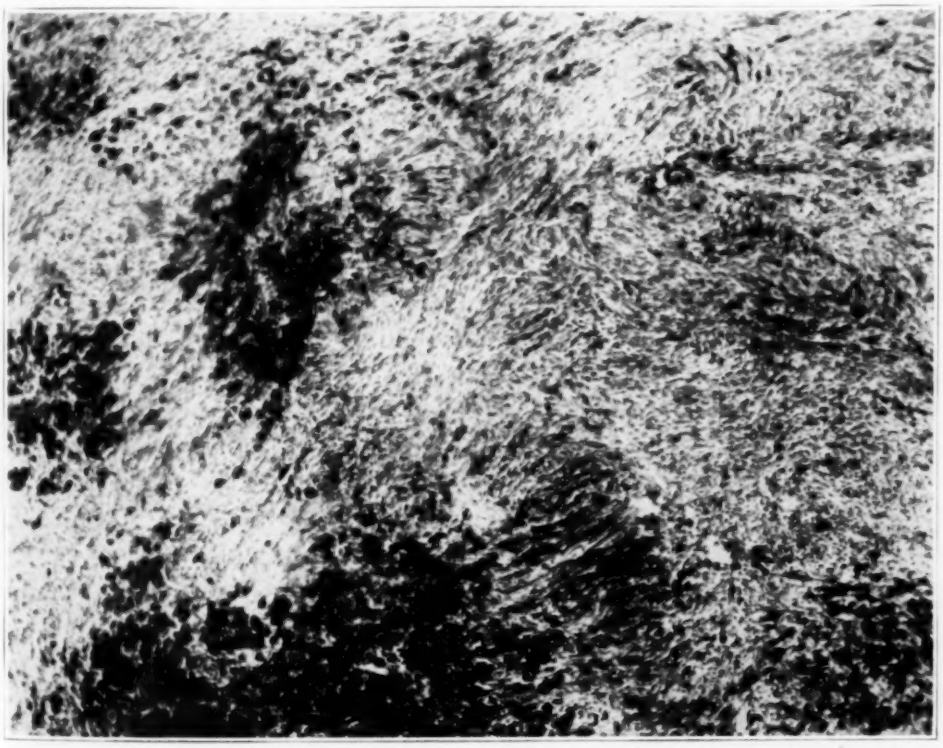

Fig. 7. (Case 2). Microphotograph. Melanotic spindle cell sarcoma of choroid. Lens A. A. Ocular 6. Bellows 78.

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DISCUSSION.

DR. GEORGE F. SUKER, Chicago: This paper of Dr. Howard's is worth a full discussion. It shows that our pathologic interpretation of certain intraocular tumors is being logically revised.

I am particularly interested in the question of pigmented and nonpigmented sarcoma. Personally, I believe there is no such thing as a genuine nonpigmented sarcoma of the choroid. They are all more or less pigmented. In infants and children, the sarcomata are but scantily pigmented, becoming more pigmented as both tumor and child grows, and most markedly pigmented after puberty.

The location of the tumor has a great bearing on the question of metastasis. If a choroidal tumor is situated near the optic disc, particularly if at the edge in a staphyloma or conus, or is near the entrance of a vena vorticosa of the choroid, or near the ciliary body, early metastasis can be looked for. There will be no metastasis in the majority of cases when the sclera is intact thruout. Early metastases also occur when rapid pigmentation takes place. This is my clinical experience.

The diagnosis of sarcoma of the choroid, I think, is not as easily made as we usually suspect. Every tumor of the choroid will produce more or less detachment of the retina, and this is not the detachment as ordinarily seen; it is a planatic detachment. Whenever you have elevation of the retina as a result of tumor, you will find at the dependent portion of the growth and retinal detachment a cystic appearance of the retina. In other words, a tumor in the eye or elsewhere being of pseudoglandular structure, a certain amount of serous exudate occurs, which is more or less translucent. Furthermore, you can aspirate with a hypodermic needle, and if you get a clear serum, you are not likely dealing with a tumor, but with a detachment of the retina; if there is a growth, you will have a sanguinous fluid, as all tumors are more or less vascular.

Transillumination in my hands has been absolutely valueless as an aid in the diagnosis of an intraocular tumor.

I believe, in order to obviate any metastasis occurring, that as much of the nerve should be taken as possible, and the earlier the enucleation, the better. However, I thoroly believe, also, that often in these sarcomas of the choroid, when metastasis does occur, it is less apt to be local than general; and strange as it may seem, I have on record and have had observation of several instances where the patient had an intraocular sarcoma and finally died of carcinoma of the liver. When enucleation is conservatively done, with a large share of the nerve incorporated, with as little damage to the vascular system as possible at the time of enucleation, when the sclera is absolutely intact and there is little proliferation of pigmented cells, then the question of longevity is very favorable. The

older the patient, the relatively less favorable the prognosis, because of a change in the character of the malignancy which takes place. What it is, I do not know, but there seems to be a change in the specificity of the socalled tumor. However, I wish to state that I believe the majority of the tumors seen in infants are of the sarcomatous type, and that they are always nonpigmented, and that the pigment is incorporated as life progresses, and particularly at the age of puberty or senescence.

Dr. William L. Benedict, Rochester, Minn.: As Dr. Howard has mentioned some statistics that were given him by Dr. Gipner of the Mayo Clinic, and has brought these out for publication, it has been necessary for us to go over these figures and correct them. A more careful analysis of the cases would give a little different indication of the occurrence of the tumor than what was inferred by Dr. Howard from the first information given him. In approximately 40,000 primary eye cases seen from 1920 to 1926, there were 28 intraocular pigmented tumors. Twenty proved to have originated in the choroid; 3 in the iris and the ciliary body; 5 of questionable origin, but probably in the choroid. In the choroidal group of 25, nine are known to have metastasized, the shortest within seven months after enucleation, or within ten months after the onset of the symptoms. The longest case was 41 months after enucleation, or 47 months after the onset. There was only one definite recurrence in the orbit six months after enucleation. Six deaths were reported from eight months to fifteen months after the onset.

Of the three tumors found in the iris or ciliary body, all were known to have metastasized; all are dead. The shortest time for metastasis was three months after enucleation and 46 months after the onset; the longest was 56 months after the enucleation. There is a history of metastasis arising 110 months after the onset of the tumor, but as this onset was five years before enucleation, it is doubtful whether the metastasis came from the tumor or originated elsewhere in the body. There is a group of nine of uncertain origin, including cases enucleated elsewhere and coming to the clinic because of recurrence. There was metastasis in five and recurrence in seven; only one metastasis without recurrence was noted. Two cases died from local extension of the tumor. The longest time without recurrence was ten years after enucleation, with death fourteen years afterwards from general metastasis. The next longest period was 24 months—2 cases. The shortest period before recurrence was five months, and the earliest death was two months after enucleation, from extension of the tumor.

This would bear out quite well the opinion of Dr. Suker regarding the likelihood of metastasis, because where the origin of the tumor was anterior, metastasis occurred early, while those originating in the middle portion did not metastasize so early. It was also found, on going over the pathologic material of these tumors, that where they metastasized early, there was a comparatively small amount of pigment. Those that were more deeply pigmented were less likely to produce metastasis or early recurrence, while those that were not pigmented grew more rapidly and metastasized earlier. This was borne out by statistics from the pathologic department regarding recurrence and metastasis of epithelioma elsewhere in the body.

DR. E. C. ELLETT, Memphis, Tenn.: I would like to say a word with regard to the difficulty of making a prognosis from microscopic sections. More than twenty years ago, I had under my care a medical man with sarcoma of the choroid. His eye was enucleated and serial sections were made of the growth. One of these sections, from which the photograph was made, passed thru a perforating vein of the sclera, and that vein, so far as the section extended, was filled with cells identical with the cells of the tumor. Apparently the portion of that vein left in the orbit was

Fig. 1. Section of a sarcoma of the choroid and overlying sclera. Vein passing thru the sclera is filled with tumor cells.

equally full of these tumor cells. That man is living and well so far as any manifestations connected with this intraocular tumor are concerned, after twenty years. Any one would have given a very bad prognosis from the examination of that section.

In none of a considerable number of these cases have I ever seen any involvement of the optic nerve—very different from the socalled glioma of the retina in infants, in which involvement of the nerve is quite common.

DR. C. NORMAN HOWARD, Warsaw, Ind. (closing): I wish to express my thanks to the discussants, and I appreciate very much the points they have brought out.

ANNULAR MALIGNANT MELANOMA (RING SARCOMA) OF THE IRIS.*

WILLIAM R. MURRAY, M.D.

MINNEAPOLIS, MINNESOTA.

Annular, malignant tumors of the iris and ciliary body are of comparatively rare occurrence. I have been able to find in medical literature seven reported cases of malignant, annular tumor of the iris, and thirteen cases of malignant, annular tumor of the ciliary body.

The following case seems worthy of report:

A farmer, aged nineteen years, entered the University Hospital December 1, 1924, complaining of loss of vision in the left eye. He stated that his parents had first noticed a change in the color of his iris in February, 1923, and a few months later he noticed that the pupil of the left eye was somewhat larger than that of the right eye, and that the pupil had gradually increased in size. He had not, however, noticed any change in vision in the left eye until three weeks before entering the hospital. There was no history of injury and there had not, at any time, been pain or discomfort in the eye.

Present Condition: In the left eye there was a mild injection of the vessels in the bulbar conjunctiva, and there was a deep injection of the ciliary vessels. The cornea was clear; the anterior chamber was very shallow; the pupil was immoble, dilated, oval, with its long axis vertical. Examination of the iris showed areas of dark pigmentation, which were apparently not raised above the level of the remaining portion of the iris. These pigmented areas extended from the ciliary portion of the iris toward the pupillary margin. They varied in size and were present, at irregular intervals, around the circumference of the iris. The iris, between the areas of pigmentation, was of normal color. There was a marked ectropion uveae present, the pigment epithelial layer of the iris presenting a marked proliferation around the entire pupillary

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margin. There were no blood vessels to be seen on the iris surface and no circumscribed tumor of the iris.

The capsule of the lens showed the presence of former posterior synechiae, there being three small, irregularly shaped, brownish pigmented spots where the pigment layer of the iris had been attached. The lens was otherwise clear. The vitreous was clear, and the retina showed no abnormality except a few small pigmented spots, which were observed along the course of the superior temporal vessels. The optic disc presented a deep glaucomatous cupping, and there was an optic nerve atrophy present. The tension was 45 mm. (Schiötz), and vision equaled perception of light. Transillumination of the eyeball was negative.

The right eye showed no pathology. Tension was normal and vision equaled 20/20.

A diagnosis of intraocular tumor with secondary glaucoma and optic nerve atrophy was made, and enucleation advised.

December 4, 1924, the left eyeball was removed, and a letter from the patient, received July, 1926, stated that there had been no recurrence of the growth nor evidence of metastasis.

The enucleated eyeball was fixed in formalin solution and sent to Doctor W. E. Camp, to whom I am indebted for the pathologic findings.

Pathologic Examination.

Macroscopic: Gross diameters of the eyeball are about normal. The cornea is clear. The anterior chamber is shallow. The pupil is dilated and oval in outline. The iris is thickened in its peripheral portion and is adherent to the cornea thruout its circumference, in the region of the angle of the anterior chamber, obliterating the filtration angle. There is a marked ectropion of the pigment epithelium of the iris at the pupillary margin. There is no circumscribed tumor to be seen in the iris, and there are no isolated nodules present. Pigmented areas are present on the surface of the iris, extending from the periphery towards the pupil margin. The lens is clear, pushed slightly forward, and has several small pigmented spots on its anterior capsule. Otherwise the lens appears macroscopically to be normal. The vitreous is clear and the choroid and retina appear to be normal. The optic nerve is deeply cupped.

Microscopic: A portion of the eyeball was embedded in

paraffin and a portion was embedded in celloidin. Serial sections, cut in a sagittal plane, were made at intervals of 10 to 15μ and stained with hematoxylin and eosin. The corneal epithelium, Bowman's membrane, corneal stroma and endothelium are normal. The conjunctival vessels at the limbus are congested, but there is no infiltration or chemosis, and there is no evidence of an epibulbar extension. The anterior chamber is shallow. The iris is considerably thickened at its root, and is adherent to the posterior surface of the cornea, at the angle of the anterior chamber, for a distance of ½ to 1½ mm. around its entire circumference. This thickening of the iris is due to an annular tumor involving the entire circumference of the iris in its peripheral half, and completely blocks the filtration angle.

The tumor is composed of round and spindle shaped cells, many of which are deeply pigmented. In places where the cells are more densely packed, polygonal cells are present and there is a tendency to alveolar formation. The nuclei are round and oval and are deeply stained. There are a few mitotic figures present. There are no giant cells, no hemorrhages or necrosis, and no perivascular arrangement of the cells. In the mass filling the filtration angle, the tumor cells are more loosely packed and extend up on the posterior surface of the corneal limbus, covering the corneal endothelium from ½ to 1 mm. beyond the line of adhesion of the iris to the posterior corneal surface. The tumor cells extend into the meshes of the pectinate ligament and the anterior portion of the ciliary body. Schlemm's canal is surrounded by tumor cells. In many places, the deeply pigmented anterior border layer of the iris can be followed thru the tumor tissue. Small tumor masses of variable size can be found scattered thruout the whole extent of the iris stroma. The major and minor iris arterial circles are surrounded by tumor cells, and there is a beginning extension into the sclera along the anterior ciliary vein.

The dilator and sphincter muscles are not involved in the tumor growth, and only at one point do the tumor cells extend to the sphincter. There is a marked ectropion of the pigment epithelium, extending around the pupil margin, and in places extending along one-half of the anterior surface of the iris. The pupillary portion of the iris is from ½ to ⅓ the thickness of the peripheral portion. There is no epibulbar extension of the growth.

The ciliary body is thinned, elongated and atrophic. The ciliary processes are normal. The anterior half of the ciliary body is invaded by dense masses of tumor cells, wedged in between the ciliary muscle fibers and the ciliary epithelium. The lens is normal except a few pigment deposits on its anterior capsule from posterior adhesions. The vitreous and choroid are normal. The retina shows postmortem changes and fragmentation from sectioning. The optic disc shows a deep glaucomatous cupping, which is filled with glial tissue, new formed connective tissue and blood cells.

Diagnosis: Annular malignant melanoma of the iris, with extension to corneal limbus and ciliary body.

Remarks.

Malignant tumor invading the circumference of the iris or of the ciliary body was described in 1898 by Ewetzky, who reported such a growth invading the ciliary body and iris, and described it as a "ring sarcoma." Vose Solomon (1882) described a case of melanosarcomatous tumor of the iris, in which the angle of the anterior chamber was infiltrated thruout the whole circle by masses of tumor cells, which passed into the meshes of the ligamentum pectinatum and thence proliferated into adjacent structures. Solomon's case is the first that I have been able to discover of that type of uveal tumor which was later described by Ewetzky as a "ring sarcoma."

Ring sarcomata of the iris and ciliary body apparently belong to the diffuse type of tumor described by Fuchs (1882), who first distinguished between circumscribed and diffuse sarcomata of the uveal tract. According to Parsons, the diffuse sarcomata differ from the circumscribed type in that they infiltrate wider areas, producing more or less thickening of the choroid, ciliary body and iris, without the formation of a definite tumor. Parsons further classifies the diffuse sarcomata into two subgroups, flat sarcomata and ring sarcomata. In ring sarcomata of the iris and ciliary body, there is usually a definite circumscribed growth combined with diffuse infiltrating extensions which produce the annular tumor, thereby differing from the flat sarcomata of the choroid, which do not occur as definite circumscribed growths.

In the above case, the iris is involved in the tumor growth thruout its entire circumference, in its peripheral portion, and

the angle of the anterior chamber is infiltrated with tumor cells thruout its entire extent, the tumor cells infiltrating the meshes of the pectinate ligament, extending along Schlemm's canal, and in places beginning to extend along the ciliary veins.

Annular sarcomata may have their origin within the iris and extend around the entire iris root and filtration angle, or they may originate within the ciliary body and thence by extension form a complete ring within the ciliary body, or the tumor may invade both the iris and ciliary body to such an extent that it may be difficult to determine the primary site of the growth.

A review of the literature shows a total of twenty published cases of annular malignant tumor of the iris or ciliary body, or of both combined. Annular tumors of the iris have been reported by Solomon[1] (1882), Hanke[2] (1898), Meyerhof[3] (1902), Alt[4] (1911), Laven[5] (1913), Li[6] (1923) and Bruner[7] (1924). Annular tumors of the ciliary body have been reported by Groenouw[8] (1899), Meyerhof[3] (1902), Parsons[9] (1903), Werner[10] (1904), Wintersteiner[11] (1907), Bergmeister[12] (1910), Alling and Knapp[13] (1912), Ischreyt[14] (1912) and Gibson[15] (1922). Annular tumors of the ciliary body and iris have been reported by Ewetzky[16] (1898), Kopetzky[17] (1901) and Pindikowski[18] (1914).

Annular tumors of the iris and ciliary body apparently are of long duration and have a tendency to grow slowly, in this respect resembling the flat sarcomata of the choroid, and also in this respect resembling the circumscribed sarcomata of the iris, which may be present for years without causing subjective symptoms. In Solomon's case, there was evidence that the tumor had existed for twenty years; in Ewetzky's case, at least eighteen years; in Kopetzky's and Pindikowski's cases, nine years. In the other reported cases, the recorded duration of the tumor was from one month to several years.

Glaucoma is a frequent complication, especially in those cases in which there is an extensive ring formation, due to blocking of the filtration angle. Intraocular tension was increased in thirteen of the reported cases, normal in four cases, minus in one case (Groenouw), and not mentioned in two cases.

A deep anterior chamber was present in five of the published cases, shallow in five cases, normal in two cases, not mentioned in eight cases. In their reported cases of ring

sarcoma of the iris, Li and Bruner refer to the similarity of the glaucoma present to that of a buphthalmos, as the anterior chamber was deeper than normal and there were no anterior synechiae present.

Pain is a variable symptom, being present in eight cases, absent in nine cases, not mentioned in three cases. In four of the nine cases in which there was no history of pain, hypertension was present.

Epibulbar extension is of frequent occurrence and may occur comparatively early. It usually occurs by extension of the tumor cells to the ligamentum pectinatum and Schlemm's canal, and along the perforating anterior ciliary vessels, especially the veins. In eleven of the reported cases, epibulbar extension of the growth is definitely described.

In fifteen of the twenty cases published, the tumor was pigmented. In the cases reported by Meyerhof (case iv), Groenouw, Parsons, Wintersteiner and Ischreyt, the tumor is described as a leucosarcoma, and in those cases of unpigmented tumors, the age of the patients varied from fourteen to seventy-two years.

The cases reported by Solomon, Hanke, Meyerhof (cases vi and vii) Alt, Laven, Li, Bruner, Ewetzky, Kopetzky, Parsons, Werner, Wintersteiner, Bergmeister, Alling and Knapp, Ischreyt and Pindikowski, are cases of complete annular sarcomata of the iris or ciliary body. In Groenouw's case, the ring formation of the tumor invaded the lower half of the ciliary body; in Meyerhof's case (iv), there was an incomplete ring formation; and in the case reported by Gibson, the lower half of the ciliary body showed an annular extension.

Li has defined ring sarcoma of the iris as a sarcoma in which the tumor extends thruout the entire circumference of the iris root and the filtration angle, forming a complete ring around it; and a ring sarcoma of the ciliary body as a sarcoma which extends thruout the whole circuit of the ciliary body, forming a complete ring within it. According to Li's definition, some of the reported cases must be classed as incomplete ring sarcomata. However, as Alling remarks, whether the ring is complete may be of little importance, for it is known that the tendency of these growths is to progress and to invade the whole anterior uveal tract.

ANNULAR MALIGNANT TUMOR OF THE IRIS AND CILIARY BODY.

	Author	Age	Sex	Symptoms	Duration	Anterior Chamber	Tn.	Annular Extension	Microscopic Examination	Epibulbar Extension
1.	Solomon 1882	43	F	Failing vision, recent pain. Noticed pin head size growth iris, increasing size.	20 yrs.	Shallow	N.	Complete in Iris	Spindle, round and globular cells. Pigmented. Angle ant. chamb. infiltrated tumor cells. Lig. pect. and Schlemm's canal invaded.	Not mentioned
2.	Hanke 1898	63	F	Vision failing 9 mos. Blind 2 mos. cataract, pain, ciliary injection, cornea hazy.	9 mos.	Shallow	+	Complete in Iris	Spindle and ploygonal cells. Aveolar. Few giant cells. Pigmented. Cellular infiltration in circumference filtration angle. (Endotheliomata).	+
3.	Meyerhof Case vi, 1902	51	M	Blindness, cataract, no pain.	?	?	+	Complete in Iris	Small, round and spindle, polygonal. Pigmented.	+
4.	Meyerhof Case vii, 1902	?	?	?	?	?	+	Complete in Iris	Spindle and polygonal cells. Pigmented.	+
5.	Alt 1911	49	F	Dim vision, pain, noticed dark spot iris no. of yrs.	No. of yrs.	Shallow in lower part	+	Complete in Iris	Spindle cells. Periphery iris thickened by tumor. Pigmented.	—
6.	Laven 1913	12	F	Ant. ciliary vessels injected. Noticed pigmented area iris, increasing size.	Several years	?	+	Complete in Iris	Spindle cells, oval and long nuclei. Pigmented.	+
7.	L[illegible] 1923	22	M	Blurred vision. Eyeball inflamed at times. Noticed several dark spots iris.	1 mo.	Deep	+	Complete in Iris	Spindle cells, round and oval nuclei. Schlemm's canal and neighboring vessels filled tumor cells. Pigmented.	—

	Author	Age	Sex	Symptoms	Duration	Anterior Chamber	Tn.	Annular Extension	Microscopic Examination	Epibulbar Extension
8.	Bruner 1924	43	M	Failing vision 5 mos., pain, ciliary injection, cornea steamy. Small brown spot iris increasing in size.	5 mos.	Deep	+	Complete in Iris	Small round cells. Entire circumference lig. pect. invaded. Pigmented (slight).	Not mentioned
9.	Murray 1926	19	M	Failing vision 3 wks., blindness, no pain. Noticed change color of iris. Pupil dilated.	22 mos.	Shallow	+	Complete in Iris	Round and spindle cells. In places polygonal and alveolar formation. Few mitotic figures. Tumor invades periphery iris and lig. pect. Pigmented.	—
10.	Ewetzky	39	F	Dim vision, rainbow colors, iridodialysis, pupil dilated, ciliary congestion, portion of cornea hazy.	18 yrs. Recurrence after iridectomy 11 yrs. before.	N	+	Complete in ciliary body and iris.	Spindle and round cells. Pigmented. (Slight).	Not mentioned
11.	Groenouw 1899	28	M	Failing vision, cataract, no pain. Noticed discolored iris.	11 wks. Injury eye 9 wks. before	Deep	—	Incomplete in ciliary body	Cells of various size and structure; polygonal, round, spindle. Large round or oval nuclei. Alveolar formation. Leucosarcoma.	+
12.	Kopetzky 1901	20	M	Pain, dim vision. Noticed brown spots iris.	9 yrs. Iridectomy sarcoma 9 yrs. before	Shallow	+	Complete in ciliary body and iris	Spindle cells; invaded filtration angle. Pigmented.	—
13.	Meyerhof Case iv 1902	72	M	Failing vision, no pain, few dilated conj. vessels. Noticed growth on iris.	1 mo.	?	N.	Incomplete in ciliary body	Small round cells, spindle cells in aveolar form near ora serrata. Leucosarcoma.	Not mentioned

	Author	Age	Sex	Symptoms	Duration	Anterior Chamber	Tn.	Annular Extension	Microscopic Examination	Epibulbar Extension
14.	Parsons 1903	14	M	Failing vision, enlargement eyeball. Ciliary staphyloma.	? Injury 7 yrs. before.	Deep	+	Complete in ciliary body	Spindle cells, large oval nuclei, meshes lig. pect. Schlemm's canal filled tumor cells. Leucosarcoma.	+
15.	Werner 1904-1905	18	M	Failing vision, noticed coal black spots iris, no pain.	9 mos.	Deep	+	Complete in ciliary body	Spindle, large round, polygonal. Alveolar formation. Pigmented.	+
16.	Wintersteiner 1907	?	F	Pain, blind 2 yrs, tumor at corneoscleral margin.	8 yrs.	?	+	Complete in ciliary body	Small cell sarcoma. Pigmented.	+
17.	Bergmeister 1910	61	M	Failing vision, no pain, iridodialysis, dark spots iris.	6 mos.	N	N.	Complete in ciliary body	Spindle and round cells. Pigmented.	—
18.	Alling and Knapp 1912	60	F	Failing vision for 2 yrs., dark spot on sclera 1 yr., pain, pigment spot on iris.	2 yrs.	Shallow	+	Complete in ciliary body	Spindle cells. Free pigment cells with large nuclei.	+
19.	Ischreyt 1912	50	M	Failing vision, no pain. Noticed small tumor at corneoscleral margin.	3 mos.	?	N.	Complete in ciliary body	Round cell sarcoma, alveolar, nonpigmented.	+
20.	Pindikowski 1914	50	F	Pain, dim vision, first noticed small pigment spots iris, increased size.	9 yrs. Iridectomy for sarcoma iris 1 yr. before.	?	+	Complete in ciliary body and iris	Spindle cells Pigmented.	+
21	Gibson 1922	45	M	Noticed growth in lower outer part of ant. chamber.	1 mo.	?	+	Incomplete in ciliary body	Spindle cells. Pigmented.	Not mentioned

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DISCUSSION.

Dr. William H. Wilder, Chicago: I think Dr. Murray has made a fine presentation of a very interesting case. I must say that I have never seen a case exactly like the one reported, the nearest being melanotic sarcoma of the ciliary body invading the root of the iris. Naturally, a tumor developing in the ciliary body might extend forward into the iris, and in such cases we can see the growth projecting forward into the angle of the anterior chamber.

Possibly the tumor reported developed from some of the unusual pigmented patches that one sometimes sees in the iris, which are probably analogous to the melanomas of the skin that the dermatologist has learned to handle with the greatest caution. It has been shown that these melanomas of the skin are very dangerous things, especially if an attempt be made to remove them, so that they are often allowed to remain by the dermatologist, even if they are disfiguring.

I note in Dr. Murray's report, that his case already showed increased tension and the signs of irritation characterized by marked ciliary injection, so I think it illustrates the second stage of intraocular growths, according to the clinical classification which was proposed by Dr. Herman Knapp. According to this classification we have: First, the stage of quiescent growth; second, the stage of irritation; third, the stage of invasion of the orbit, and finally, the stage of metastasis. It is very important to consider these stages when making our estimates of the prognosis in any of these cases. If we are fortunate enough to have discovered the growth before the stage of irritation has begun, the prognostic chances are considerably greater for a favorable outcome. If, however, the case has already entered the stage of irritation, with marked ciliary injection

and especially with increased tension, the gravity is considerably increased. When the tumor has extended to the third stage and is already growing outside of the sclera, the chances for favorable results are far less, and, of course, when metastasis has occurred the prognosis would be fatal.

It is rather important, it seems to me, in connection with this matter of the extension of the growth backward, to make careful section of the optic nerve and to have a long piece of it to examine. I notice in the discussion of the subject of melanotic sarcoma of the choroid, that Dr. Suker stated it was not frequent to see the optic nerve invaded by the tumor. I should like to dissent from this, because I have seen a number of cases that showed distinct invasion of the optic nerve with the pigmented cells.

Dr. W. E. Camp, Minneapolis, showed several lantern slides.

Dr. William R. Murray, Minneapolis (closing): This case has been designated as a malignant melanoma of the iris. The term "malignant melanoma" has been used to avoid confusion in classifying it. Whether or not it belongs to the epithelioma type is a question which seems to be debatable.

The type of tumor here is the usual type of sarcoma found in the iris. The interest in this case lies in the annular extension of the growth, involving the root of the iris in its entire circumference and the angle of the anterior chamber. Why it is that this growth took this particular form is questionable. It is quite possible that it is due to the histologic and anatomic structure of the root of the iris and the angle of the anterior chamber. It may be due to the loose arrangement of the tissue in the peripheral portion of the iris, or it may be due to the circular distribution of the blood vessels in the root of the iris, which favors the advancement of tumor cells along these lines. It may also be due to the dissemination of the tumor cells thru the action of the aqueous humor, which may carry cells into the angle of the anterior chamber and deposit them, where they undergo proliferation and thence extend around the angle of the anterior chamber.

TRANSACTIONS

OF THE

THIRTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTO-LARYNGOLOGIC DIVISION

SCARLET FEVER STATISTICS.

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Scarlet fever as a frequent cause of partial or total deafness is to be regretted. Ellison Ross,[1] in reviewing his summary of the literature on scarlet fever as a cause of chronic otitis media, gives us this table:

SCARLET FEVER AS A CAUSE OF CHRONIC OTITIS MEDIA.

Authors	No. Cases Otitis Media	No. Cases Due Scarlet Fever	% Otitis Due Scarlet Fever
May-Schmalz	2,500	137	5.50
May-Yearsley	544	27	5.00
May-Burkhardt			
Marian	1,950	85	4.33
May	5,613 D.M.	572	10.18
Bezold	623	89	14.30
Bezold	714	103	14.40
Sprague-Blau et al.	14,045	55	3.90
Weil-Burkhardt	4,309	488	11.30

He concludes "that approximately 4 to 15% of all cases of chronic otitis media are due to scarlet fever." He gives May's report on "5,613 deaf mutes, and the cases are not purely otitis media. It is striking to note that 10% of these thousands of cases of complete deafness was due to scarlet fever."

A few years ago, Cook Co. Hospital placed on their scarlet fever service men competent to examine ears, which was done routinely every four hours. Early paracentesis was their order, not waiting for a rise in temperature or a complaint of pain in the ear. In their series, they had one mastoid operation and dismissed no dicharging ear.

STANDARD TAKEN.

We have taken as our standard, that every patient developing an ear complication shall go out not only with a dry

ear and good hearing, but an intact tympanic membrane. If we fail with any patient, we wish to know why we fail.

Strict asepsis, early paracentesis under a local anesthetic which also disinfects the drum and canal; ether drops twice a day following paracentesis is the rule. When the patient enters, the ears are examined by the interne, and usually by the resident physician. After that, examinations are made if the patient has prolonged temperature, rise in temperature, or complaint of pain, or the house staff has time for routine examinations. Ear clinic is held twice a week by the attending staff, consultations are willingly given, operations are done by otologists especially interested in mastoid work.

The scarlet fever ward is a potential school in conservation of hearing. The aseptic methods of a contagious hospital are important in the teaching of both nurses and internes, and probably more vital in the outcome of an ear than usually recognized. The nursing staff is quick to learn the importance of the ear condition. Many of the members go out to public health work, where they will have influence. Internes study early changes in the tympanic membrane, and perform both postmortem and clinical paracenteses.

Scarlet Fever Statistics, Los Angeles General Hospital, Jan. 15, 1925 to July 15, 1926.

Scarlet Fever Alone.

One patient (F. N.) was admitted with acute mastoiditis, the 28th day of scarlet fever, for a simple mastoid operation, which was done immediately.

Two patients with chronic otitis media entered with scarlet fever. One (Pott's disease) was returned to the Orthopedic Hospital, with request his ear have attention; the other, previous bilat. mastoid operations released to enter the general ear clinic for radical mastoid operations.

Of 473 cases, 58 patients, or 12.25%, developed ear complications.

Two of these died; otitis was not the cause of death.

Two required mastoid operations (one bilateral), due to continued discharge.

With but one exception (congenital lues), the other 54 left hospital with healed tympanic membrane, hearing good.

SCARLET FEVER WITH DIPHTHERIA.

One patient entered with discharging ear; was operated upon later for intermittent discharge.

Of 18 cases, 7 patients, or 38.88%, developed ear complications.

Three left the hospital without consent.

Two had mastoid operation as emergency procedure.

One had mastoid operation for continued discharge.

One left with tympanic membrane healed, hearing good.

In these two groups, we find that there were 66 patients entering with tympanic membrane intact who developed otitis media. One of these, or 15 plus %, had bilateral failure to heal spontaneously. Eight, or 12%, had monolateral failure. We offer the records of these nine with our own criticism.

GROUP I. LEFT HOSPITAL WITHOUT CONSENT OF EAR STAFF.

R. M., age 2½ yrs., entered the 7th day of scarlet fever and diphtheria. The 39th day of his illness, he complained of pain in L. E.; paracentesis was done. The following day, spontaneous rupture of the R. E. occurred. The 65th day of his illness he left the hospital to be quarantined at home. A year and a quarter later, R. E. discharging, T. & A. operation again refused; mastoid operation not considered by parents.

Criticism: Failure to lance R. E. Failure to secure parents' consent for operation. Failure to impress health officer with danger of home quarantine, patient's sister contracting scarlet fever, with bilateral chronic otitis, deafness resulting.

R. C., age 21 yrs., entered the 5th day of scarlet fever; complained of pain in R. E. the 13th day of her illness; myringotomy was done the 14th day. Examination by the tuberculosis staff gave no evidence of disease in lungs. Detre test very slightly positive. Wassermann test negative, but history of congenital lues given by family physician, the 74th day of her illness, when she left the hospital; slight discharge present in R. E.

Criticism: Delayed paracentesis. Failure to elicit history of lues, and institute specific treatment. Mastoid operation should have been considered.

B. A., age 6 yrs., entered the 4th day of scarlet fever and diphtheria. Bilateral paracentesis was done the 5th day of

her illness. She left on the 65th day of her illness, the L. E. healed, the R. E. discharging. Examination the 5th month, the R. E. discharging. Tonsil and adenoid operation advised but refused.

Critiscism: Failure to lance R. E. before complaint of pain. Necessity of T. & A. operation with mastoid operation later should have been anticipated, and parents' consent gained.

E. M., age 7 yrs., entered the 3rd day of scarlet fever and diphtheria. Bilateral paracentesis was done on the day of entrance. The tuberculosis staff reported no evidence of disease of lungs. Some tracheobronchial glandular enlargement, but probably not tubercular. Detre test essentially negative. The L. E. healed, the R. E. was discharging on day of leaving hospital, the 91st day of his illness. T. & A. operation refused by parents; mastoid drainage not considered by parents.

Examination 8 months later: Scanty but dirty discharge present in canal, perforation has increased in size until only margin present. Referred to general ear clinic again. T. & A. operation urged.

Criticism: Necessity of T. & A. operation with mastoid operation later should have been anticipated, and parents' consent gained.

Group II. Operated Upon for Acute Mastoiditis

R. G., age 6 yrs., entered, the day of scarlet fever and diphtheria not known. The 3rd day, L. E. bulging, myringotomy. The 6th day, R. E. bulging, mygringotomy, which later healed spontaneously. The 20th day, L. mastoid tender, temp. 104°, simple mastoidectomy. Pus exuded under pressure when cortex was opened, pneumatic cells filled with pus and granulations. Dismissed the 61st day, with mastoid wound and M. T. healed. After a week at home he had a cold, L. E. discharging; on the 8th day, his aunt developed scarlet fever, possibly due to contagion from his ear. Three months after mastoid operation, wound was opened; tonsils and adenoids removed on account of recurrent sore throat. A month later "no drainage, small opening in post. wound."

Criticism: Failure to lance ears before bulging occurred. Failure to remove tonsils and adenoids before dismissal from contagious ward.

M. F., age 8 yrs., entered the 14th day of scarlet fever and diphtheria. Rise in temp. 18th day, bulging L. drum, myringotomy. Paresis of the L. facial nerve appeared on the 36th day. Simple mastoidectomy (left), mastoid containing pus under pressure; abscess cavity extending to antrum; neither sinus nor dura exposed. Facial paresis disappeared by the 30th day after operation; patient was dismissed the 67th day of her illness with tympanic membrane and mastoid wound healed. Subsequently, lymph node post. to mastoid incision swollen, and was opened thru old wound.

Criticism: Ear should have been lanced before bulging occurred.

GROUP III. OPERATED UPON FOR CONTINUED DISCHARGE.

M. T., age 2½ yrs., entered the 14th day of scarlet fever. Spontaneous rupture R. E. the 15th day; of the L. E. the 23rd day of her illness. Examination by tuberculosis staff: no evidence of tuberculosis. Child cried during examination. Bilateral mastoid operation the 80th day of her illness. Simple mastoidectomy, bilateral. Rt. cortex perforated; mastoid cells necrotic and filled with granulation tissue and pus. Lt. cortex intact. All cells necrotic and filled with granulation tissue and pus. Lateral sinus uncovered. Mastoid wounds kept open with difficulty, but ears continued to drain. Left on the 130th day of her illness, with the consent, but against the desire of the ear staff.

Criticism: Failure to perform paracentesis. Failure to warn house staff to keep mastoid wounds well open. Failure to remove tonsils and adenoids.

E. P., age 11 yrs., entered the 1st day of scarlet fever; the R. drum was bulging, the L. congested. Myringotomy was done on the R. E., paracentesis on the L. E. Tonsils and adenoids removed on the 27th day of illness. The L. tympanic membrane healed, but R. E. continued to discharge. Tuberculosis staff reported chest negative for tuberculosis. The 35th day, mastoidectomy (Rt.); periosteum slightly adherent; cortext intact; mastoid cells filled with granulation tissue; a polyp about the size of pea in the mastoid cavity; pus in mastoid antrum. Dismissed the 79th day, large perforation, ear dry.

Criticism: Delayed paracentesis in R. E.

A. G., age 3 yrs., entered the 4th day of scarlet fever and diphtheria. Paracentesis of L. E. the 12th day, spontaneous recovery. Paracentesis of R. E. the 18th day. Examination by tuberculosis staff, report negative. Tonsils and adenoids removed a month before the mastoid operation (R), which was done the 109th day of her illness. Modified radical operation (R). Large pneumatic type, filled with granulation tissue, pus and granulations in tympanic cavity. Necrotic bone and incus removed. Healing prompt; dismissed the 132nd day of her hospital residence.

Criticism: Failure to lance R. E. before complaint of pain. Improvement in general condition following T. & A. operation, demonstrated it should have been done earlier.

GROUP IV. OPERATED UPON FOR ACUTE MASTOIDITIS.*

F. N., age 4 yrs. R. E. pained the 5th day of scarlet fever, was lanced by attending physician. Entered contagious hospital the 28th day for mastoid operation, ear discharging, swelling over mastoid, moderate rise in temperature. Simple mastoidectomy Rt.; cortex perforated; subperiosteal abscess; acellular type of mastoid; pus and granulation tissue in mastoid antrum. Patient dismissed the 27th day following operation. Wound healed, ear dry but perforation present, hearing good.

OPERATED UPON FOR INTERMITTENT DISCHARGE.*

H. R., age 4 yrs., entered the 15th day of combined scarlet fever and diphtheria, with R. E. discharging. Parents state ears discharged at 6 wks. of age. T. & A. operation at 13 mos. by Dr. Baum of Denver, on account of discharging ears. Report from tuberculosis staff: "Strongly positive Detre test. Slight tracheobronchial glandular involvement, but no activity in the lungs present." The 82nd day of his illness, or 67th day in the hospital, simple mastoidectomy was done. Rt. cortex intact; cells filled with granulation tissue; necrosis around mastoid antrum. Dismissed 18th day after operation, ear dry, perforation present. Four mos. later, drum healed, no evidence of old perforation, hearing normal.

*Both entered on account of ear complication, hence not included with the preceding nine.

Conclusions.

Early paracentesis, with strict asepsis following as well as at time of paracentesis, lessens the seriousness of ear complication.

Diligent attention by the staff to the ear problem in scarlet fever increases the confidence of the health officer and the pediatrician. Their influence is vital—the health officer in restraining those with discharging ears from going home to spread infection; the pediatrician in choosing the time when the patient is best fitted for mastoid operation.

Consent of parents to a mastoid operation as an emergency procedure is easily gained; but consent to an operation to cure a discharging ear, when the patient has recovered from a critical illness, is difficult. Anticipation of this period by the ear staff develops the interest of the parent concerning the child's ears and hearing. Consent for operation is then given when needed.

Reporting of these ear cases to the school principals or teachers should be as much a personal matter as is possible. They should be asked that the patient having had an ear complication be under observation for evidence of a cold, and be sent home; that he be excused from attendance in severe weather; that any evidence of recurrent otitis or lessening of hearing receive proper attention immediately.

Segregation of scarlet fever patients in the modern contagion hospital, with better attention to the ear complication than we have herein reported will definitely reduce ear pathology.

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FURTHER OBSERVATIONS OF MASTOID INFECTIONS IN INFANTS.*

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ST. LOUIS, MISSOURI.

Prior to the last few years, a diagnosis, in infants, of a surgical mastoiditis was rarely made except upon the basis of external signs—redness, swelling or subperiosteal abscess. We now know that an infant may have a very severe mastoid infection, capable of producing profound constitutional symptoms and even death, without any of the external signs being present.[1]

In adults, one of the most dangerous types of mastoiditis runs its entire course, often leading to serious intracranial or constitutional complications and death, without presenting any external evidence of the disease. This has been called the hemorrhagic type of mastoiditis, because the local dissemination of the infection takes place by way of the blood vessels instead of by contiguity, as occurs in the coalescent type of the disease.

Each of these types of mastoid infection has its analogue in the infant, differing from the adult condition only because of the anatomic difference between the adult and the infantile temporal bone.

The tympanic cavity and ossicles of the new born baby are practically the same as that of the adult in size and shape, with the difference, however, that in the baby the entire upper part of the cavity is filled with soft myxomatous tissue folds, which prevent direct communication between the hypotympanum and the aditus ad antrum. This mucosa, when infected, is subject to very rapid purulent transformation, so that epitympanic suppurations are more common in infants than in adults, and adequate drainage of this portion of the ear cavity by myringotomy is more difficult to maintain.

The Eustachian tube of the new born is relatively shorter, more horizontal in position and larger in lumen than that of

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the adult. This difference furnishes the explanation for the fact, that a baby may have a tympanic cavity full of purulent secretion, and yet inspection of the drum reveals neither redness nor bulging. Both of these symptoms, which are practically always to be found in the presence of an acute ear suppuration in the adult, are the results of pressure. In the baby, however, the pressure in the middle ear is relieved by spontaneous drainage of the ear abscess thru the relatively larger and shorter Eustachian tube. Alexander[2] states that the tympanic membrane of the new born is thicker and more resistant than that of the adult, and gives this as one of the reasons for the length of time often required for the ear drum of a baby to rupture spontaneously in a middle ear suppuration.

My own experience would lead me to doubt this statement, and I believe that the failure of the infant's drum to rupture is due more often to the release of pressure by the evacuation of the purulent contents of the middle ear by way of the Eustachian tube. This observation has been further confirmed by the fact, that by means of the nasopharyngoscope, a stream of pus coming from the pharyngeal orifice of the tube has been observed in several babies whose ear drums were intact, showed no bulge or redness, and incision of which revealed free pus in the tympanic cavity.

The type of mastoiditis which, in infants, gives rise to a subperiosteal abscess, is a distinct and characteristic clinical entity. It usually follows rapidly upon a typical acute suppurative otitis media, and is accompanied by pain, some fever, and as a rule a low leucocytosis. Severe constitutional symptoms are rare in this form of the disease, altho occasionally there is a moderate amount of toxemia present until the abscess ruptures thru the cortex. With the appearance of the external signs, the fever, pain and constitutional manifestations subside rapidly, and it is not uncommon to see a little patient with a tremendous subperiosteal abscess of several days duration with no pain, fever or other discomfort manifested at this time. Following operation, these cases almost without exception make a rapid and uncomplicated convalescence, and usually the functional return of the ear to normal is complete

The pathology in this form of the disease is in every way analagous to the coalescent mastoiditis of adults. The infection of the middle ear thru the semiopen tube, the involvement of the mesodermal folds in the epitympanic cavity and mastoid

antrum, with partial liquefaction, swelling and obstruction in the narrow aditus, is rapidly followed by an increase of pressure in the mastoid antrum, with capillary thrombosis, death of bone cells and rupture in the line of least resistance, which is usually the thin lateral wall of the antrum. During the stage of pressure, pain, fever and malaise are all present, but with the external perforation thru the bony cortex and the release of the pressure, the subjective symptoms as a rule subside promptly.

Until the last few years, this has been considered the typical clinical picture of mastoiditis in an infant, and in cases where the principle objective features of this symptom complex were absent, a diagnosis of mastoid infection has only rarely been made.

Largely as a result of postmortem examinations on infants who had died of gastrointestinal disease, we have learned that there is a type of mastoid infection in the baby that is apparent only to the trained observer, because in most of the cases, none of these objective features of the disease are present. The only signs which are of otologic significance are the changed color of the drum and a distinct downward sag of the superior canal wall just external to the annulus. In this form of the disease, the constitutional evidences of absorption are the predominating clinical features. This syndrome in a baby has been commonly called "cholera infantum," and is abrupt in onset, usually requiring early and vigorous supportive measures to prevent a fatal termination before the source of the infection can be ascertained and removed.

Jeans[3] describes the pediatric features of this condition by saying, "The symptoms, in general, consist of a marked and rapid loss of weight, fever, diarrhea, dehydration, and the appearance of intoxication. The intoxication is characterized by drowsiness or stupor and pallor with a grayish hue; it may appear simultaneously with the fever, or follow it in a few hours. The temperature curve is inconstant. When fever existed previously, a secondary increase may occur. Diarrhea usually parallels and coincides with the dehydration and weight loss. Sometimes the onset of diarrhea is slightly delayed, and in a few otherwise typical cases, diarrhea has been absent. Usually there are from eight to twenty mucus containing, foul smelling stools daily. The dehydration is reflected in the weight loss and the loss of skin elasticity: it is sometimes of such marked degree as to cause the appearance

of extreme emaciation. Prior to the establishment of drainage from the site of infection, parenterally administered fluid is often reflected in an increase in the diarrhea. The leucocyte count has varied from 13,000 to 30,000 or more. Vomiting may occur thruout the course of the illness, with varying degrees of severity. Refusal of food is a fairly constant feature. Marked degrees of acidosis, as measured by the carbon dioxid combining power of the plasma, have been noted. The urine may at times contain casts and slight amounts of albumin; glucose has not been found."

The pathology in this type of the infection is not due to pressure, but is that of a rapid and massive absorption of toxic products, which act on the mucosa of the gastrointestinal tract of the infant probably by damage to the capillaries.[4] Due to the fact that the bony elements which later go to make up the temporal bone are but loosely connected in the infant, the absorptive properties of the infantile middle ear are unquestionable greater than in the adult. The fact, that in thirty-five cases of subperiosteal abscess in infants coming to operation at the Saint Louis Children's Hospital in the last six years, the majority of the cultures showed a staphylococcus or nonhemolytic streptococcus, while the cultures from the ears of thirty-two babies with the other form of the disease almost without exception gave a hemolytic streptococcus, would suggest that there may exist something of bacterial specificity for each form of the disease.

The chief reason for the failure, in the past, of otologists to take cognizance of this type of infection in the baby, has been that these cases are always seen first by the pediatrist, who, if he does not appreciate the intimate connection that often exists between obscure ear infections and gastrointestinal disease in the infant, fails to call the otologist into consultation. Merriott, Jeans and Floyd and others have repeatedly called attention in pediatric literature to this association, and now each year we are seeing more and more of these cases which previously practically all died. The proper recognition by pediatrists of the significance in the baby of this disease combination, and the closer study by otologists of infections in the infantile ear will, in a few years, result in an understanding of the pathology and treatment, and perhaps prophylaxis of this condition, that will surely bring about an-

other material contribution to the reduction in infant mortality.

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DISCUSSION OF LEFLER'S AND ALDEN'S PAPERS.

Dr. Fred J. Pratt, Minneapolis: The records of the contagious cases at the Minneapolis General Hospital for the period, January 1, 1925, to July 1, 1926, were checked over by Dr. Gerald M. Koepcke, an intern in the hospital.

The number of cases are as follows: Scarlet fever, 771; diphtheria, 701; smallpox, 296; measles, 205; chicken pox, 52; and erysipelas, 84; making a total of 2,109 cases.

Out of this number, 17 cases developed acute otitis media; ten cases followed scarlet fever, five cases measles, one case pharyngeal diphtheria, one case bronchial pneumonia. Ten of these cases were children under fourteen years of age and seven were adults.

Eighteen cases developed acute suppurative otitis media; twelve cases followed scarlet fever, four measles, and two scarlet fever and measles. Ten of these cases were children under fourteen years of age and eight were adults.

Seven cases developed acute otitis media followed by mastoiditis, all complications of scarlet fever. Six were children under fourteen and one was an adult.

Twelve cases entered the hospital with acute mastoiditis, eight with scarlet fever, three with measles and one with diphtheria. Seven were children under fourteen years of age and five were adults.

All cases of mastoiditis were operated and the suppurative cases put on the dry treatment.

Out of 771 cases of scarlet fever, 27 developed middle ear suppuration, with 15 mastoid operations.

I am unable to state the final outcome, as all cases leave the hospital as soon as possible, tho an attempt is made to follow them in the dispensary. As this is the only contagious hospital in the city, many cases are private and of course would be hard to follow.

Dr. L. E. Brown, Akron, O.: Both of the essayists are to be congratulated on the papers presented by them, not only from the educational standpoint, but for their practicability.

In discussing mastoiditis in young children as a complication of scarlet fever, where the diagnosis is made before the typical symptoms and signs, such as the postauricular swelling, tenderness, pain, redness, etc., are

present, we find that it practically brings us to the study of atypical cases, because it is some atypical condition that exists, anatomically or otherwise, in these cases which prevents the usual symptoms and signs from appearing. Consequently, one must bear in mind many things which contribute to such conditions, and it is for this reason that one learns to anticipate the unexpected in mastoid diagnosis.

The following facts should be borne in mind: First, the very small percentage of cases of scarlet fever occurring in infants. Second, that when the infant is so afflicted, the possibility of an ear infection is much reduced by the inability of so young a patient to blow the nose and thus force the infection from the nasopharynx into the middle ear; also by the attention that is given to the nose and throat of those patients by the careful pediatrician of today. Third, that when infection does occur in the middle ear, it is the result of coughing, sneezing or vomiting, thus forcing the infection thru a short, patulous Eustachian tube into the middle ear, where it usually can be controlled if proper attention is given to the nose, throat and nasopharynx, accompanied by early and free incision of the membrana tympani, thus preventing mastoid involvement. Fourth, it is a definite fact that the systemic manifestations are of the greatest importance in young infants, for the reason that young children are not as sensitive to pain, and their attention can be attracted in other things, hence it is very difficult to recognize the usual pain and tenderness of a mastoid involvement. Fifth, the typical sagging of the posterior superior canal wall, unless very marked with almost complete occlusion of the canal, is very difficult to make out. And, as is well known, in the very young infant there is no bony canal wall to sag, thus almost eliminating this valuable sign.

Hence, the signs and symptoms which one must rely upon to make their diagnosis in these very young children are as follows:

(a) The X-ray findings, taking both sides for comparison.

(b) Blood culture and blood count, especially in reference to the percentage of polynuclear cells.

(c) Condition of the pulse and temperature, especially where these remain high, in spite of free drainage from the middle ear, following a well established convalescence from the exanthema and after eliminating a coexisting nephritis or suppurative pyelitis, etc.

(d) Pulsation of the opening in the membrana tympani is to be considered very important, indicating the retention of infection behind the drum membrane.

(e) If the patient has been observed closely, it will be noted that he has had a decided chill.

(f) As Kerrison of New York has very aptly stated it, that indefinable something in the patient's general appearance or condition, which in spite of temperature, mastoid tenderness or other typical signs will indicate or at least suggest favorable progress in one case and unfavorable in another. This is particularly true in the case of infants and young children.

Thus we realize that the constitutional manifestations are of the greatest importance, and naturally will be more marked in the child in

which the abscess did not rupture externally, due to the much thicker and more sclerotic cortex, thus causing a decided absorption of the toxins, than where this did occur, giving vent to the pus and preventing further systemic contamination.

In reference to the most opportune time to operate a mastoiditis in infants and young children, especially following scarlet fever, I think is best illustrated by a recent report by S. T. Sorenson of New York, which by the way substantiates other reports of a like nature, and is as follows: In a series of 57 cases of acute mastoiditis in children following scarlet fever, all except three were operated upon after the development of the subperiosteal abscess. All of these socalled delayed cases were opened, drained and made an uneventful and complete recovery. The other three were operated very early, before the development of the subperiosteal abscess, with 100 per cent. fatalities.

This signifies one thing, that as the condition progresses and develops the subperiosteal abscess, which is usually following the active stage of the scarlet fever, the patient who has probably had rather a stormy experience is given an opportunity to develop a certain immunity to this infection, and will withstand the shock of a mastoid operation much better at this time than earlier.

It is a well known fact, that in the past, as the essayist has stated, the percentage of early diagnosis in infants was small. This has been due either to oversight, or lack of knowledge on the part of the general practitioner or the pediatrician to recognize the necessity for very close observation, noting every point in the child's condition, and not attributing symptoms which he cannot account for to teething, worms, colic, etc. He must also realize that there is considerable difference between the signs and symptoms presented by the older child and those presented by the young infant, and it is only when these gentlemen begin to recognize these facts and thoroly acquaint themselves with above mentioned points necessary to make a diagnosis in infants, or are willing to consult with a competent otologist, thus making it possible for infants suffering from a mastoiditis subsequent to scarlet fever to receive prompt and proper care, that the oversights mentioned by the essayist will be eliminated.

Dr. Harold I. Lillie, Rochester, Minn.: What Dr. Lefler has said about careful observation and early opening of the drum being a conservative and important measure, is well worth consideration. I prefer incision or myringotomy to paracentesis, as paracentesis suggests merely a stab wound in the drum. Incision and myringotomy is as easily performed and is more effective in facilitating good drainage.

Dr. Alden has brought up a very important subject. There must be enthusiasts and conservatives as advocates of any question. I believe Dr. Alden does not intend to have the impression go out, that in all babies with gastroenteric disturbances, this type of mastoiditis is a causative factor. My experience has been confined to twelve cases. There was not sufficient evidence of involvement of the ear to warrant exploration of the mastoid, and all the patients recovered under general treatment. The incidence of mastoiditis as a causative factor must be small. Otologists are not all ready to accept the proposition on its face value. Careful judgment must be exercised in choosing the cases for operation.

In laboratory animals, pus and secretion found in cavities may show bacteria on culture, but it may be part of general infection and not the causative factor per se.

Dr. John F. Barnhill, Indianapolis: The one thing most worth while shown by the case reports is the good effect of early treatment. The report adds another proof to the rather well accepted fact, that early free incision of the drumhead in acute infectious diseases of the middle ear is not only the best treatment for the middle ear affection, but is also the best prophylaxis against unfortunate consequences—possible impairment of hearing and possible mastoiditis.

Dr. Lefler has given a clear statement of the progress of middle ear suppuration under favorable environments. One feature of the report is somewhat surprising, in that she was able to dismiss every case with dry ears, since it is not always possible to cure aural discharge after scarlet fever. Often the discharge becomes chronic despite our best efforts, and not infrequently does not cease until after a radical mastoid operation has been performed. I take it that the cases here reported were those following a mild type of scarlatina.

Dr. Alden's paper is valuable, and I appreciate the reports he has made of his rather extensive investigation of the subject. One part of the present paper which interested me was that in which he describes secondary intestinal infection. The question arises as to why children with but slight aural discharge should have secondary intestinal complication. It is a common experience that both children and adults who have sinus disease or nasopharyngeal infection swallow large quantities of mucopus or pus daily without setting up intestinal disease. It is believed that the gastric juice of normal persons will sterilize and render innocuous almost any quantity of pus the patient may swallow. I would, therefore, ask Dr. Alden if, in these little children, the gastric juice is incapable of sterilizing the small quantity of pus which may pass down the Eustachian tube and is swallowed, or if the infection of the intestine is carried to the intestine thru the blood stream?

Dr. Virgil McCarty, Kansas City, Mo.: Since the original work of Dr. Alden and Dr. Lamb was published, we have, in the past two summers, at the Children's Hospital in Kansas City, instituted a routine otologic examination of all children with summer complaint, or summer diarrhea or gastrointestinal infection.

The thing I want to ask Dr. Alden is to give a little more clearly his method of diagnosis. I confess, that after the examinations of several hundred children, I did not know whether they had ear trouble or not. I find extreme difficulty in determining whether or not there exists an ear infection. I have done a good many paracenteses on these infants, or incision if you will, and found a dry ear. However, I have found four cases of discharging ears during this time, and I have had the temerity to do a double mastoidectomy with very spectacular results on the diarrhea in three of them. The diarrhea cleared up almost immediately with the cessation of the ear discharge. I am still plugging away at this, with a good deal of enthusiastic support from Dr. Berger of the pediatric staff.

Dr. A. A. Hayden, Chicago: I think the importance of the entire scarlet fever situation has been greatly increased for the otolaryngologist

since the various serum treatments for scarlet fever have come into use. What I mean is this: In looking over Dr. George Dick's statistics in the Durand Hospital for Children's Diseases in Chicago, since his serum has been used there, I returned to this conclusion, that the toxemia noticed in the ordinary scarlet fever case is due to the scarlet fever only for the first seven or eight days. After that, it is due to some other source of infection in another part of the body, preferably the nose and the accessory nasal sinuses, occasionally to pyelitis, but generally in our own field of work. With the use of the Dick serum, the toxemia of scarlet fever itself, pure and simple, can be controlled. Inasmuch as the majority of the other foci are in the nose and accessory nasal sinuses, including the mastoid, is it not in the otolaryngologist's power to control the sequelæ which usually result in the death of the patient, where a fatal termination takes place? It simply means that these cases must be diagnosed early, and must be treated early and radically when the diagnosis is established.

Another thing that struck me very favorably in the way of prevention, was the importance of the use of suction. These children nearly all have noses filled with pus. As Kerrison has shown in his book, the floor of the nose is considerably higher in the child than the tympanic cavity. In other words, the pus in the nose of an infant has a better chance to run into the ear and tympanic cavity. That, it seems to me, accentuates the advisability of keeping the nose free from pus, not by irrigation, not by blowing, but by simple suction.

Dr. Anna B. Lefler, Los Angeles, Calif. (closing): I wish to thank you for the interest you have taken, and for the response there has been in discussion.

Dr. Carmody has asked regarding the nasal infection in these patients; we have been unable to give sufficient time and work to the problem to warrant a statement.

The Cook County Hospital report has been most valuable and stimulating to us, and I regret that we could not give credit to the man who published it. We wrote the Cook County Hospital contagious division, but received no answer.

With reference to Dr. Lillie's comments about paracentesis versus myringotomy, we encourage the resident and intern to do a paracentesis on scarlet fever patients when the slightest change occurs in the appearance of the drum. We encourage them to do this to prevent congestion of the drum. We have had four different residents in eighteen months, and I think each of them believes that bulging of the drum in scarlet fever is a very serious thing; that paracentesis must occur before bulging takes place. Myringotomy is preferred if bulging has occurred.

Tomorrow Dr. Daniels will give us work of value in the dietary care. Our patients were fortunately cared for in their diets. Every scarlet fever patient is put on a nephritic diet when he enters, and cod liver oil is given in all cases having otitis.

Dr. Barnhill, you will find each case record states whether or not the perforation was healed.

Dr. Arthur M. Alden, St. Louis, (closing): In closing I wish to thank all the gentlemen who discussed my paper, including those who so

thoroly disagreed with me. In answering some of the questions that were brought out in discussion, I shall take first Dr. McCarty's question as to diagnosis.

At the St. Louis Children's Hospital, every child with gastrointestinal disease of this particular type is given a careful daily ear examination, not with the head mirror alone, because this does not furnish sufficient illumination to determine what has taken place in the fundus of an infant's ear canal. We have found, that for this purpose, the electric otoscope which supplies brilliant illumination and some magnification is indispensable. Whenever the ear of a baby who is suffering from diarrhea, vomiting and anhydremia shows any deviation from the normal in color, luster or position, it is promptly opened. These incisions are often difficult to keep open, and may require repeated reopenings in order to maintain drainage until the condition subsides. We have often seen marked improvement in the general symptoms of a child follow this type of myringotomy. None of these children are operated upon until it is definitely determined that the ear condition cannot be eradicated by more conservative measures. The indications for operation are based upon pediatric, not otologic, considerations. When, in spite of repeated reincisions, the sag in the superior wall persists, the temperature remains high and the weight curve shows a distinct downward trend, then we realize that more radical means must be used to check the condition or disaster will be the result.

Dr. Barnhill asked the question as to how the infection gets from the ear to the gastrointestinal tract. It is not the result of swallowing the ear secretions, but by direct absorption of the toxic products from the middle ear into the blood and lymph stream, and the systemic dissemination of these toxins, which apparently have a specific action on the capillaries of the gastrointestinal tract of the baby.

Dr. Carmody's question relative to the presence of sinus disease in this same gastrointestinal syndrome is a timely one. Jean and Dean have shown, in a paper presented this last year, that this same intestinal symptom complex in a baby may be the result of a sinus infection as well as a mastoiditis.

MINIMIZING THE TERROR OF AN OPERATION.

(With Lantern Slides).

SECORD H. LARGE, M.D.

CLEVELAND, OHIO.

One of our large manufacturers remarked to me, that after observing the doctors employed in his plant, he had come to the conclusion that doctors become calloused and are not concerned about the pain which the patient suffers.

Those about to have an operation should be put at mental ease to eliminate, as far as possible, whatever apprehension may be present. Special attention is necessary for individual cases. In some, a very simple procedure, such as washing out an antrum or even an ordinary routine examination, is a great ordeal. What would be a minor operation for one person becomes a major for another. In using instruments, such as the nasal and ear specula, roughness should be avoided. Small details, such as warming the ear speculum, make for the patient's comfort.

The more nervous the patient, the greater are the pains taken to have the nervous system quiet. A patient may have had one or more operations, and be so wrought up, that when another is necessary, unless special precautions are taken, the shock may be so great that a long time will elapse before the patient entirely recovers. If, shortly after an operation in which there has been considerable shock, a complication necessitating another operation arises, the utmost care should be taken so that the nerve reserve is not exhausted. Thus the nervous system may be likened to an electric battery, which will stand a great amount up to a certain danger point, but beyond this, any further demands on it will produce considerable and, sometimes, even irreparable damage.

Many things which seem of minor importance to the surgeon are really of great magnitude to the patient; for example, the opening of a stitch abscess, the removal of packing, Michel clamps and adhesive plaster, the passage of Eustachian catheters and bougies.

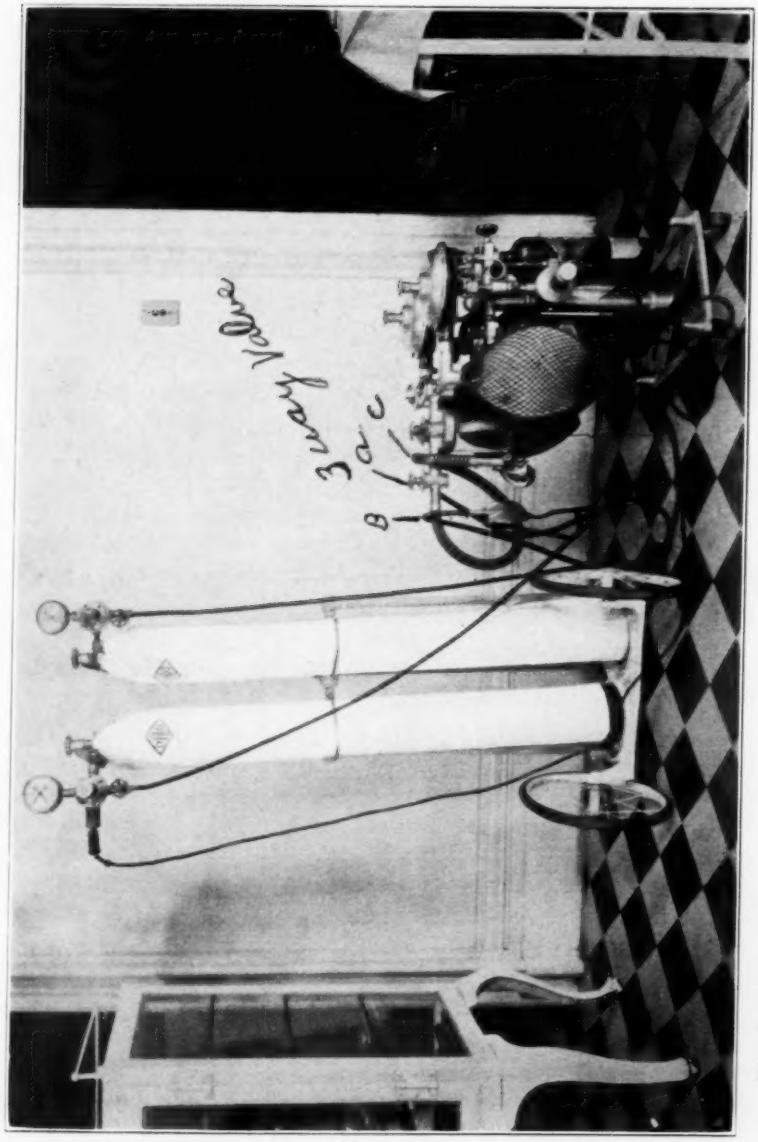

Fig. 1. Gas machine with attachments. (a) Three-way valve controls gas to nose, mouth, or both.

Goitre cases require special attention. We have as a patient a boy who had a simple goitre, together with a chronic tonsillitis and neuritis. Naturally, a tonsillectomy was advised. He went to another surgeon, and had the operation under local anesthesia. Shortly afterwards, he returned to our office with all the symptoms of a toxic goitre. He relates that the operation was so painful that he collapsed. We feel that the shock brought on his toxic goitre. In all toxic goitre cases, we refuse to do any operation until the thyroid condition is dealt with either surgically or medically.

Children require special handling. If you tell a child that you will not hurt him and then do so, he will never forget it, and any subsequent operating or treating will be rendered very difficult.

To combat undue nervous excitement, several drugs have been used, as morphin and scopolamin, either separately or in combination, pantopon and the bromides. Only the bromides can be given over any long period. They exert a very quieting effect. Our method is to have adults take fifteen grains of sodium bromid three or four times a day, for four or five days before the operation. To do away with its unpleasant taste, we have it made up with fluid extract of glycyrrhiza. Another form of bromid which we have found useful is Sedobrol. Each cube contains the equivalent of fifteen grains of sodium bromid. Three cubes in a teacup of hot water and one-third of a grain of pantopon, hypodermically, are given before operations under local. We prefer pantopon to the other opiates, because it has less tendency to produce nausea or excitement, and it does not tie up the secretions. Sedobrol is quite palatable, tasting very much like beef tea. It is very beneficial after local tonsillectomies.

Children are given 1/200 grain of scopolamin by mouth.

We no longer use twilight sleep, because with it some patients become very excited and others quite cyanotic.

One of the greatest causes of nervous shock is rough surgery. Perhaps a great deal of this is due to haste. How often have we heard it said that this or that surgeon was a good operator, but rough. I think that, as a rule, the longer a man is in practice, the more sympathetic and gentle he becomes. Teachers in medical schools should impress upon the students the necessity of gentleness. Because of the delicate manipulation required in ophthalmic surgery, I value highly my five years experience in ophthalmology.

Fig. 2. Blue sheet with complete contents of sterile pack.

Fig. 3. Blue sheet in position and spring mouth prop.

Fig. 4. Beginning gas anesthesia with attachments in place pertaining to nasal operation, when gas is administered entirely thru mouth.

Fig. 5. Three such tampons are placed in the nonoperated nares; one each in the inferior, middle and superior meatus.

We are careful not to have one patient entering the operating room while another is being brought from it. We try to have the instruments kept from the sight of the patient. During the operation, the entire face of the patient is covered with a blue sterile sheet, except for a small opening for the operative field.

All of our operations are performed with the patient in the reclining position, even during the application of the cocain and the injection of the procain solutions. Needless to say, it is more comfortable and safer. Care is taken to inject the

Fig. 6. Tonsillectomy under local and gas analgesia.

procain solution slowly, because of its rapid absorption, which often produces considerable apprehension. We are of the opinion that the use of too strong solutions of adrenalin is dangerous and is sometimes fatal. We are very sparing in the use of cocain and adrenalin, no solution of the latter stronger than 1:80,000 being used. In nose operations, strengths of 1:200,000 is the rule. Apprehension, fear and syncope are combated much better by whisky by mouth than by any of the other stimulants such as strychnin, aromatic spirits of ammonia, etc.

Practically all of our operations in adults on the nose and throat, and even on the mastoid, are performed under local anesthesia. In certain cases, where the patients are somewhat

apprehensive, gas analgesia is used in conjunction with local anesthesia. Operations on the nose by this combined method were impossible until we worked out the simple apparatus which I will show you by lantern slides. With children, and with those who are extremely nervous, we prefer general anesthesia. In such cases in adults, we favor local anesthesia combined with general anesthesia, as brought out by Crile and which he has called anoci-association. Those patients having an ether anesthetic are given nitrous oxid and oxygen induction.

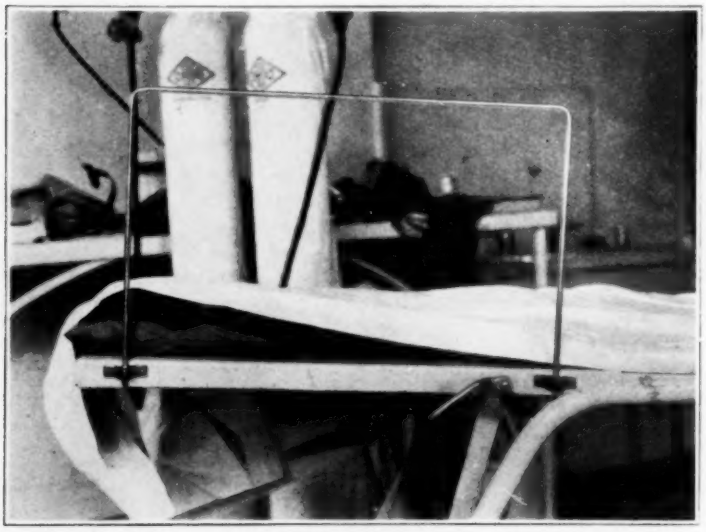

Fig. 7. Rod attachment for arrangement of sheets in mastoid operation.

Another general anesthetic which we have used over 5,000 times without a fatality is ethyl chlorid. In 1905, together with Dr. Edgar D. Brown of the Pharmacological Laboratory of Western Reserve University, I carried out a series of experiments with it on a number of dogs. We found that it was not necessary to abolish the reflexes in order to operate on these dogs without sensibility to pain. Under light anesthesia, various operations could be performed without any evidence of pain. This, also, holds true in children and adults. It is used extensively in the British Isles, having been employed in the Royal Infirmary at Edinburgh alone for over 23,000 tonsillectomies. For its administration, we had a special apparatus made, slides of which I will show later. This apparatus has

no advantage over the simple method of using a mask made of a newspaper and a towel, or the ordinary open mask.

The greatest use we have found for ethyl chlorid is in performing paracenteses in the home. It is useful, also, in opening a stitch abscess, or in doing painful dressings, etc. We always use it as a local anesthetic previous to subcutaneous injections. It has many advantages, as ease of transportation, the simplicity with which it may be administered, freedom from irritation of the mucous membrane, quick recovery,

Fig. 8. Showing arrangement of sheets in mastoid operation.

absence of nausea, and the fact that the operation can be performed under light narcosis.

We practically never do a paracentesis without a general anesthetic. If in the office, nitrous oxid and oxygen are used; if in the home, ethyl chlorid. We have as a patient, a boy of ten, who has had nine paracenteses. The first four were done without any anesthetic. When he came to us for the fifth, he was almost uncontrollable. Since then he has not objected to any of the subsequent ones.

It has been our rule, when applying gauze next to a wound, or inserting packing into a mastoid cavity, or placing tampons

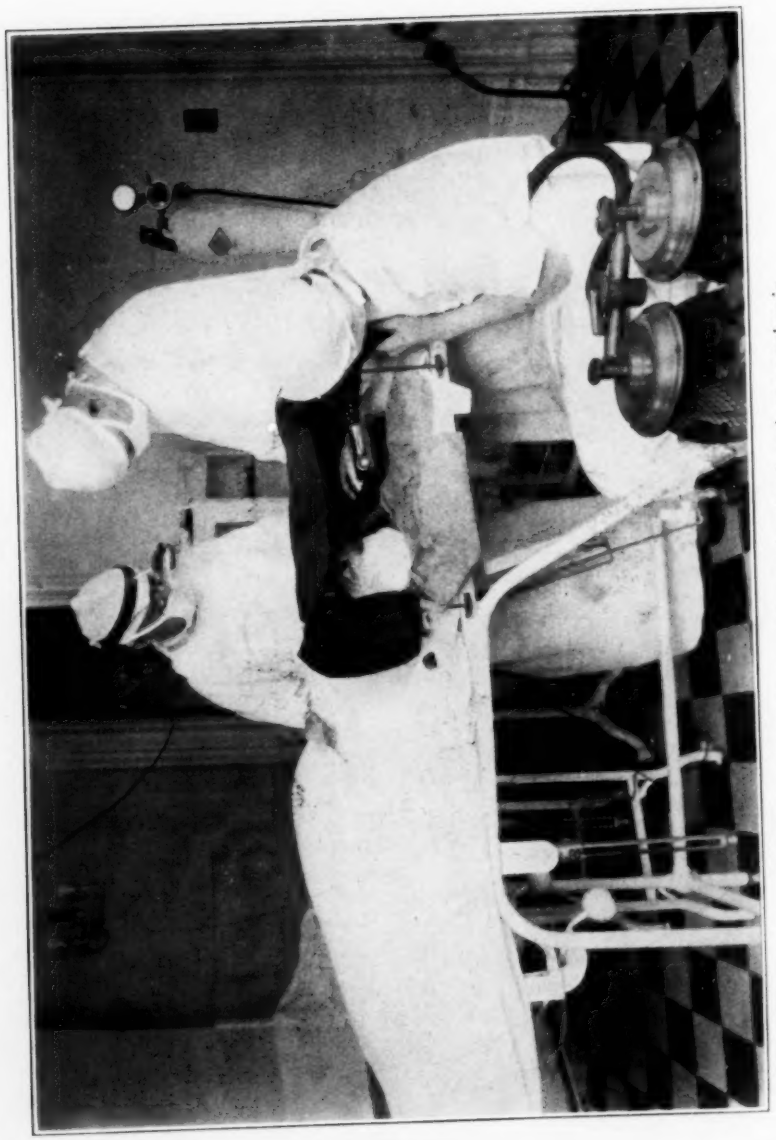

Fig. 9. Mastoid operation under local and gas analgesia.

in the nose, to have them well covered with sterile vaselin. This facilitates and makes painless their removal. If they tend to stick, particularly the nasal tampons, they can be loosened with hydrogen peroxid.

Care is taken in the insertion of Michel clamps so that they are not squeezed too tightly, because this makes their removal difficult and painful.

Fig. 10. Field of operation entirely separate from anesthetist.

Before we introduce a bougie into a strictured Eustachian tube, we dip the end of the bougie into sterile vaselin, and then into cocain flakes. It is then slowly passed into the tube and is practically painless.

Our whole object, then, is to quiet the patient before operation, remove all apprehension during the operation, and keep him comfortable by freedom from pain afterward. In this way the dread and fear of the operation will be minimized.

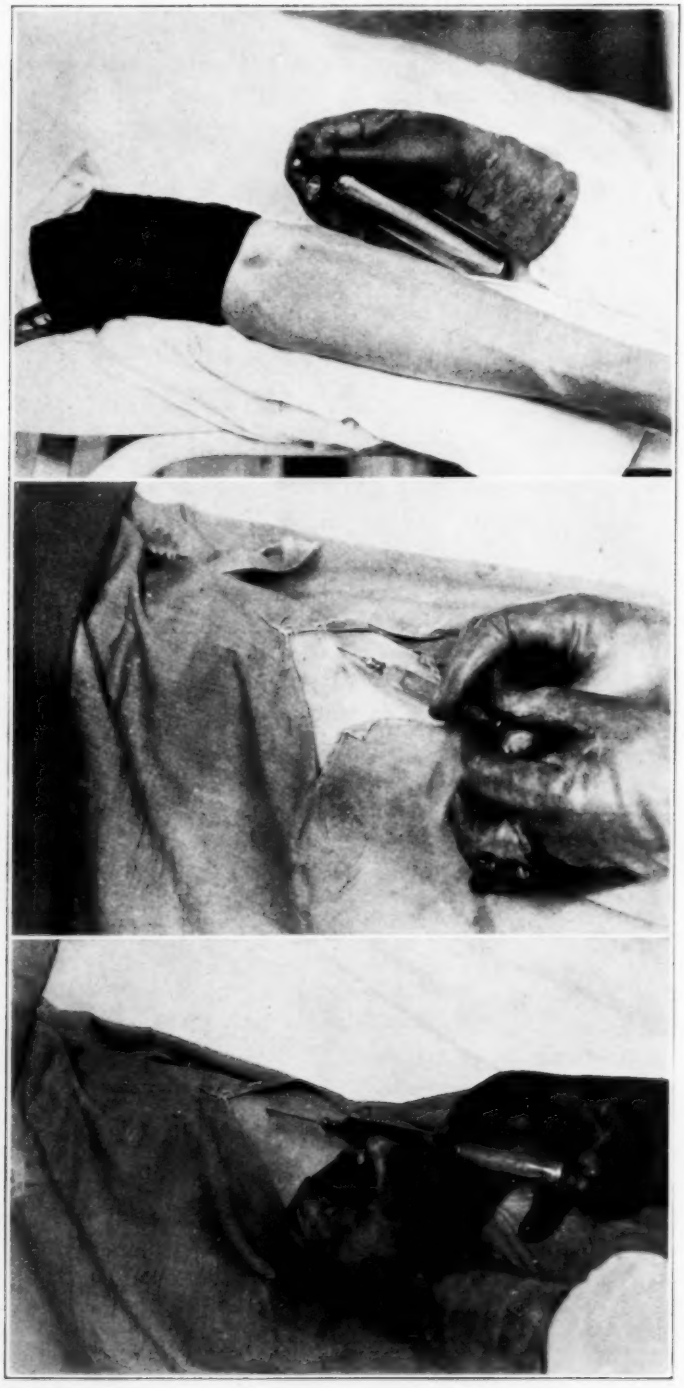

Fig. 11. Painless method for intravenous injection, and spinal puncture. (a) Ethyl chlorid over vein. (b) Small bleb of one percent novocain. (c) Injection thru anesthetized area.

DISCUSSION.

Dr. W. Albert Cook, Tulsa, Okla.: I do not think the medical profession are getting careless. I think it is the whole general trend of things, that everything is going at a greater rate of speed than ever before. Nowadays a great many doctors take a history negligently, simply because they are in a hurry to get thru their work and get to the golf course. We did not use to have that to contend with.

The nervous patients must be satisfied from the time you get them under your care until you make up your mind to operate, or if they are not, you will be better off if you do not operate, as they will never be satisfied. The doctor says he carries candy to give to the children. We give them chewing gum—it is cheaper and has the same effect. Children must be handled diplomatically at any rate. We had a case about two weeks ago, a child with a penetrating wound of the eyeball, with some of the iris protruding, and it was necessary to do an iridectomy. With a couple of drops of cocain, I was able to do the operation without an anesthetic. I use general anesthesia for all tonsil operations in children and in adults of the nervous type. But take a person who is moderately nervous, when we use local anesthetics we usually give HMC a half-hour before the local anesthetic, and in that way we can operate on a very nervous person—the nerves of the patient are quieted down as well as the throat.

The doctor speaks about the minimizing of the dread of operation that should be taught in the medical schools. I think the best place to learn that is in the general practice of medicine. I think every one taking up a specialty should be encouraged to go into the general practice of medicine for two to five years, because there he comes in contact with the people and gets something he cannot get in any other way. In cataract work, it is a good idea to take these patients and introduce a speculum and instruct them a little—get them to look to the right and the left, up and down, and get them trained beforehand, so they will know what to expect when they do get to the operating table.

Dr. Frank L. Dennis, Colorado Springs: Dr. Large has stressed a very important point, and that is the matter of making operations, if not pleasant, certainly not so terrifying as a great many people are apt to think. I think we doctors as a rule do not realize the attitude of the patient towards operations. We tell a patient that we will open his ear drum, or take out tonsils, or operate on the nose, or whatever it may be, and it seems a very simple matter to us: but the way the patient looks at this thing is very different. I think this way of preparing the patient for operation is perhaps one of the most important steps that could be taken. Of course the patient must have confidence in the doctor. You have often heard a doctor say, "I believe this man would let me cut his head off." That shows the confidence of the patient in the doctor—a very necessary thing.

So far as drugs before operation are concerned, we use very little. In some nasal operations, we use twilight sleep, or partial twilight sleep. In some special cases, we give a little morphin, but not as a routine.

In handling a child, your first interview is the most important one. If you establish cordial relations at that time, subsequent interviews will

be simpler and easier. With regard to giving children mild anesthesia, like somnoform and nitrous oxid, that is all right, but we must remember that some children fear the approach of the mask as much as anything else.

Dr. Large spoke of the nasal packing, and, as I understood, he uses vaselin soaked gauze. The matter of packing is one thing that the laryngologist has been trying to get away from in recent years. We all remember the way we used to keep patients' noses packed, and how much more pain they felt on removal of the gauze than at the operation. I think the profession as a whole is getting away from this packing, altho in certain conditions it is essential, and in those cases we use a Cargile membrane on a Simpson splint, which makes the removal of this packing a simple and painless procedure.

Dr. William V. Mullin, Cleveland: Dr. Large's paper appeals to me tremendously, and I do not think its value can be expressed. But I do not agree with Dr. Cook. I think that we all are getting careless, and that it comes from overwork and fatigue and having too much to do. If you analyze it, you will find that on the days you are tired and overworked, you are quite likely to handle patients in a little different way than when you feel fresh. Another thing is that the doctors themselves are not very good sports about operations. They do not like to have them done, especially under local anesthesia, and very often they postpone having operations that they would advise their patients to have done. We sometimes think patients are joking when they speak of their horror of instruments and so forth, but this terror is really genuine, and I believe the more it is minimized, the more attractive those operations which are a necessity will be. Dr. Large referred to Dr. Crile's method in operation for hyperthyroidism. Dr. Crile has for years felt just this same way as Dr. Large does, and that is that the patients must be protected from all kinds of shock. He does all his thyroid work in the room, and the patients do not know when their operation is to be done. If the patient has a special nurse, some other nurse is apt to come in and give them morning care, so that they are not suspicious if strangers come into the room. He will not leave the patient alone for one minute. Also, if they cannot get to the operation right away, the anesthetist will start a light analgesia, talking to the patient all the time. That leads up to another point that Dr. Large has mentioned, in which he is fortunate, and that is that he has his own hospital and his own corps of workers, who have been with him for a long time and who, therefore, are trained in his technic. It is more difficult to get this in a general hospital; it is not easy to get the nurses and house men and orderlies to get this point of view. If you are not continually after them, you will find them slipping out of those things which you intended to have carried out.

It is an advantage, especially with children, in having a woman anesthetist. At the Cleveland Clinic Hospital, the anesthetist is there at the operation under local anesthesia just the same as if the patient were completely under. They are with the patient all the time, they are talking to him; they are ready to let him know, that if there is the least bit of distress, he may have analgesia. Seldom does he ever get beyond the analgesic stage for a thyroid operation. I think there is a great advantage in having the cooperation of a trained and competent anesthetist.

DR. HARRY A. SMITH, Delta, Colo.: In minimizing the terror of operation, there are two steps. The first step is psychologic. If you sit down with the patient before you, there is an exact psychologic time when you can tell the patient what the operation consists of, that it is dangerous, and you do not shock them. The time depends partly on the patient and partly on yourself. Then, when the patient returns, he is prepared mentally, and that is as much a factor as drug preparation. A great deal of pain can be inflicted on the patient, mentally prepared, without any anesthetic and he will not complain.

The next step is drugs. With a nervous patient you must use drugs, but as some one has said, there is not such a great danger there, because they are already nervous; the danger is that you make a well patient nervous. If a patient has a good nervous balance, you may make him neurotic, but the neurotic cannot be made much worse.

DR. FREDERICK F. TEAL, Lincoln, Neb.: I do not think there is anything that a patient enjoys more than the fact that doctors occasionally are operated on. If you want to prove this, be operated on yourself. It will cause you to be more thoughtful of your patients than anything I know of.

Frequently after the injection of novocain for tonsil operation, there will be a lot of nervous excitement. The patient will say the operation did not hurt, but that his heart threatened to hop out of his chest. It is our custom now, immediately before operation on the tonsils under novocain, to give the patient about 15 minims of aromatic spirits of ammonia, which will quiet a lot of that nervous excitement. Occasionally the patient will apparently go into a faint or syncope. It really is not, but he gets pale and almost unconscious. The best thing we have found for that is a few whiffs of ether. It is easily applied and does not take much time. I do not know of any better stimulant for cocain than ether.

The removal of dressings following nasal operations is made much easier if you use the method Dr. Large speaks of. Before putting in your vaselin gauze dressing, simply put a couple of strips of narrow gauze, well greased, against the septum and then pack the nose. You can subsequently remove the dressing down to the two short pieces you put in on the septum, and leave those if necessary. If they stick, leave them in. The main dressing you can usually remove without any discomfort.

DR. RAY K. DAILY, Houston, Tex.: I have always laid great stress on consideration for the patient's feelings—both physical and psychic. I think that the brutality with which some surgeons control their patient's behavior on the operating table—seen more often in Europe than in this country—is not only unnecessary, but is detrimental to the patient's well being.

Dr. Large is at a great advantage in having his own hospital and nurses. It is the careless remarks and incompetent service of improperly trained nurses that fill the patient with fears for his recovery and suspicion of the treatment that is given him. Most of us have to do our work in a general hospital, where often nurses are assigned to the specialist's operating room for their first surgical experience.

One point that I find very valuable in keeping patients quiet, while operating under local anesthesia, is that of carrying on a conversation with them continually; if their attention can be diverted from themselves to

some subject that interests them, and they are assured at intervals that they are doing well and everything is proceeding smoothly, that tense feeling of anxiety and impending danger is done away with.

For nasal packing we use gauze saturated with compound tinctur of benzoin; it can be left in the nose longer than any other packing I know of, and need not be removed till it is saturated with secretions, when the removal is painless. In radical frontal and maxillary sinus operations, we often leave it in for a week.

I can not agree with Dr. Large on the necessity of general anesthesia for myringotomy. We have been doing them for years, and I am convinced that given a child who has not been previously terrorized, and giving the local anesthetic enough time to act, a myringotomy can be performed without the child knowing it.

Dr. H. B. Lemere, Omaha: The classification of subjects for local anesthesia into sympatheticotonics and vagotonics, will give us some indication of their vasomotor reaction and mental attitude toward operative procedures. The shaking, the fear and the fainting of the vagotonic type, is often an interfering factor in the operation. Recently it occurred to me that caffein-sodium-benzoat might help to eliminate the fainting and apprehension in this class of cases, and I have been using a hypodermic injection of 7½ grains, half an hour before operation, with the happiest results.

Dr. Secord H. Large, Cleveland (closing): Dr. Cook did not understand me. We always give a general anesthetic to children. For the last few years we have been using Pantopon rather than HMC., inasmuch as it is more efficient and causes less excitement. We have discontinued the use of morphin except in rare instances.

Success in using local anesthesia combined with gas analgesia depends to a large extent on the anesthetist. The confidence of the patient must be secured, any apprehension or excitement allayed, and the patient's mind should be occupied by something other than the operation. The good anesthetist can obtain all this by engaging the patient in conversation dealing with something in which the patient is interested. This conversation can be carried on thruout the entire operation. This need not interfere with the operator, as he is entirely separate from the anesthetist by the arrangement of the sterile sheets as shown in the lantern slides.

LOCAL ANESTHESIA IN EXTERNAL OPERATIONS ON THE NASAL ACCESSORY SINUSES.

ROBIN HARRIS, M.D., and W. LIKELY SIMPSON, M.D.

MEMPHIS, TENNESSEE.

The principal object of this paper is to present for your consideration a routine for external operations on the nasal accessory sinuses, principally the frontals, the ethmoids and the sphenoids. This procedure has been developed by gradually weeding out the little details found bad, and holding on to and adding such as were thought good. Careful attention to matters which might be, and often are, lightly passed, has proven of much value in final results.

The preliminary preparation of the patient for such an undertaking as radical operation on the frontals, the ethmoid and the sphenoid of one side, under local anesthesia, is most important. As to medication, one of two regular plans are employed. In one hospital, where only male patients are handled, ¼ grain of morphin sulphat and 1/200 grain of hyoscin hydrobromid are given in 2 c. c. of 25% magnesium solution 1½ hours before operating time, and ⅛ grain of morph. sulphat in the same solution 15 minutes before the patient leaves for the operating room. At other hospitals, the patient is given morphin sulphat, grain 1/8, in 2 c. c. of 25% solution magnesium sulphat one and one-half hours before the operation. In 20 minutes after first dose, morphin sulphat, grains 1/8, and atropin sulphat, grains 1/200, are given in 2 c. c. of 25% solution magnesium sulphat. A third dose, the same as in the first, is given 20 minutes after the second.

With either plan, the patient is placed in a darkened room two hours before operation. A tactful nurse is given charge. No visitors are allowed. The patient is not allowed to talk, and can occasionally be persuaded to keep the eyes closed. The nurses' job is an important one, but they do not fail you if time is taken beforehand to explain just what is necessary and why. When the stretcher arrives, the patient is lifted on to it, and a towel is placed over the eyes. The operating room

is ready, no instruments are in sight, but the patient's eyes are kept covered, and he or she is gently and quietly lifted onto the operating table. If quiet is had the first 20 or 25 minutes, ordinary conversation by the surgeon and assistants is not noticed by the patient. Occasionally the patient sleeps at intervals during the operation.

The whole face is painted with 5% solution of mercurochrome, and sterile towels are placed so that the breathing is free and easy. The inside of the nose is swabbed with a mixture of adrenalin chlorid solution, 1:1000, one part, and 4% cocain hydrochlorid, two parts; then with 1% mercurochrome solution. Then the side of the nose to be operated on is packed with a strip of gauze wet with the above described cocain-adrenalin solution.

Twenty c. c. of ½ of 1% novocain solution is placed in a medicine glass. Twelve drops of fresh adrenalin chlorid solution, 1:1000, is added. Care is taken to inject no other than this solution; 15 c. c. is usually injected. Special attention is paid to injection of the lacrimal sac region and supraorbital nerve region. Injection is made all along the line of expected incision, that is, along the margin of the orbit and the side of the nose. The incision may be made six or eight minutes after the injection is finished. The bleeding will be surprisingly little, one or two or three bleeding points usually have to be caught, and occasionally require tying. For ordinary chronic pansinusitis, the Lynch incision and technic is used. In elevating the periosteum, injection of one or two places may be, but usually is not, necessary. The size of the frontals is very well determined by X-ray before the operation. The frontal is opened with gauge and mallet. The opening is enlarged with a Citelli rongeur or heavy Hajek punch. Very few patients complain when the frontal sinus is cleaned out with Coakley curettes and sharp malleable ring curettes. If any pain is felt, packing with cocain-adrenalin solution will stop it. When the ethmoids are opened, there will be pain unless packing and working are alternated. Work a bit, then pack, and the pain will be nil. Then, too, the bleeding is so well controlled, that the landmarks and field are in plain view before the operator at all times. The nasal nerve, where it makes its exit from the cranial cavity, is accompanied by blood vessels. These are clamped and tied on the orbital side, then cut internal to the clamp. The whole operative and operated field is free of

Figure 1. A patient draped, ready for injection.

Figure 2. The two cardinal points of injection.

Figure 3. Skiagram showing frontal lobe abscess.

blood when the operation is ended. The time consumed is less than when general anesthesia is used. No postnasal plug is necessary. Usually the patient does not expectorate during the whole procedure. If desired, the contents of the mouth are easily removed by means of suction. The absolute control of bleeding is a joy and a satisfaction. In closing the wound, no catgut sutures are used in the subcutaneous tissue. The skin is closed with dermal. Drainage is by iodiform gauze into the nose. Stitch abscesses are rare—especially has this been so since the use of catgut subcutaneously was discontinued. Occasionally it becomes necessary to make an external opera-

Figure 4. The same patient, side view, frontal lobe abscess.

tion for an acute suppurative abscess. The anesthesia, however, is usually not so complete as when there is no swelling. One such case was a rupture of the ethmoids into the orbit, and the pain was severe. The patient was a young woman, 8 months pregnant. Incision was as above under local anesthesia, establishing drainage externally and into the nose by means of rubber tubes; relief was had at once. In 24 days a full term normal baby was born.

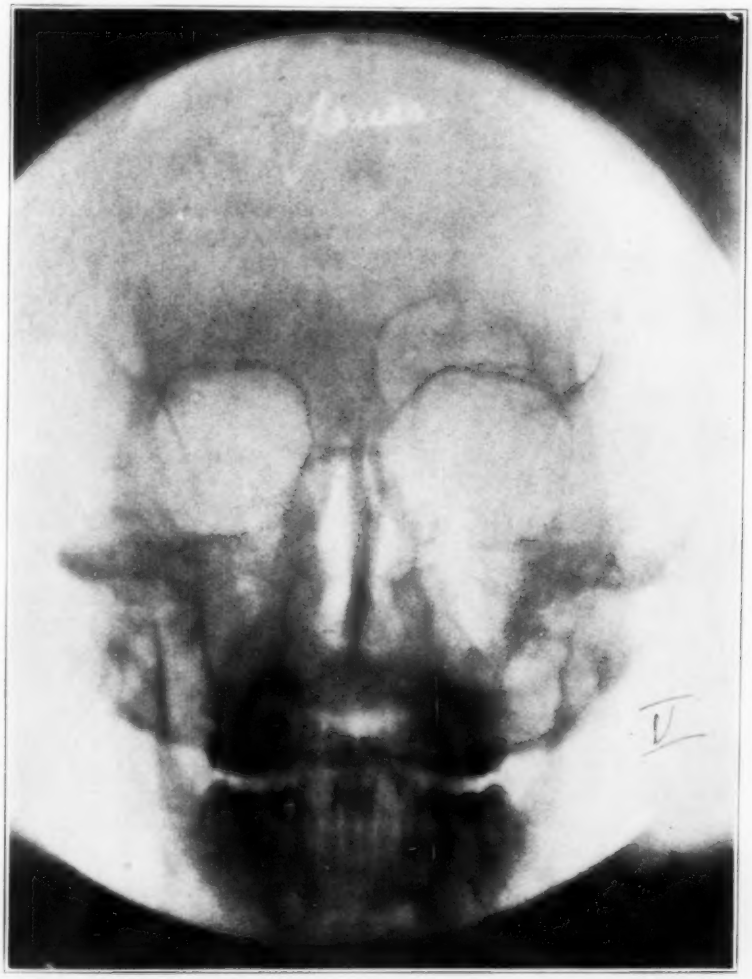

Figure 5. Chronic pansinusitis of one side. Complete loss of vision from optic nerve atrophy. External operation was too late to save vision.

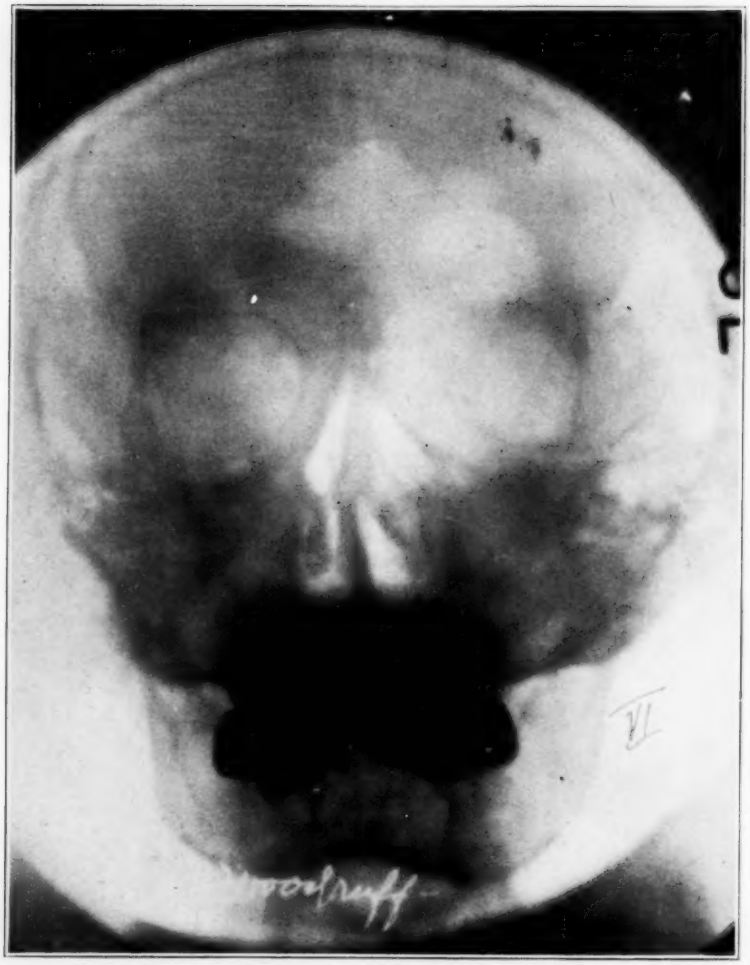

Figure 6. Skiagram showing chronic bilateral pansinusitis. Killian four years previously on the large frontal sinus. External operation on right side, small frontal, according to the method of Lynch, under local anesthesia. Later, the left side had to be reopened because of persistent left sided headache. The orbital roof was completely taken away at this time and all of the ethmoid cells removed.

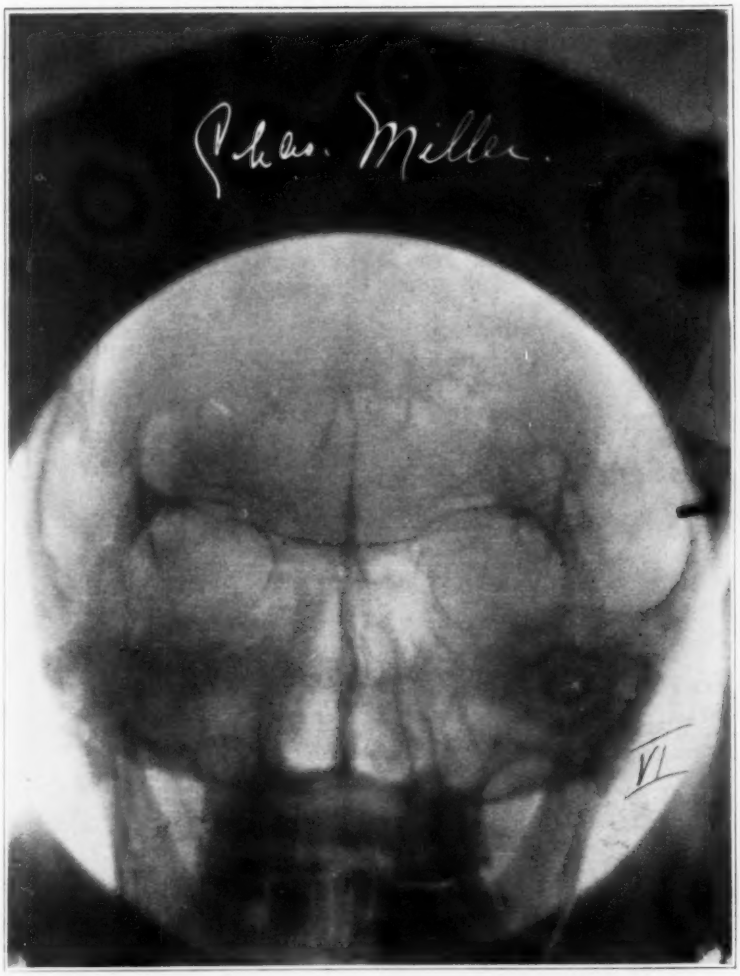

Figure 7. Skiagram. Chronic bilateral pansinusitis; sinuses very large External operation both sides. Good results.

Among other cases may be mentioned an osteoma of the septum between the frontal sinuses. This was a chronic pansinusitis. The sinuses were operated on as above indicated, after the tumor was removed.

Another was a large frontal lobe abscess. The roentgenologist was so sure the posterior wall of the frontal was ruptured, that the frontal was opened. No rupture was found

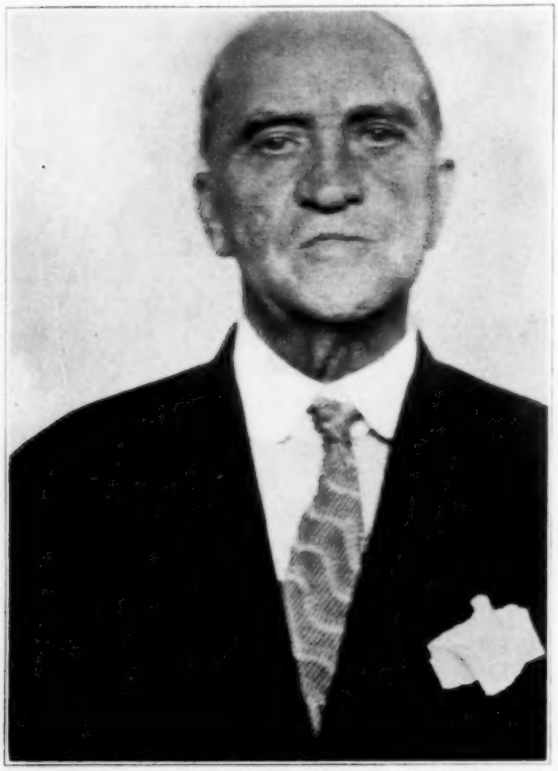

Figure 8. Photograph after bilateral operation as described.

The posterior bony wall of the sinus was partially removed, and the frontal lobe abscess drained thru this opening. The patient made an uneventful recovery.

While this open method is perhaps a bit more precise, a bit more exact, than the usual intranasal methods, the results are so universally good, that either general or local anesthesia

Figure 9. Photograph after unilateral operation. This was an acute rupture of the ethmoids into the orbit. Drainage tubes were used externally for ten days.

may be employed, according to indications in the particular case or the preference of the operator; yet, forty-eight such operations under general anesthesia, and forty-six under local anesthesia have gradually worked us into local anesthesia as a routine, and rarely now do we find it necessary to recommend an operation under general anesthesia.

Figure 10. Photograph six months after external operation for chronic pansinusitis. This patient has gained much weight.

DISCUSSION.

Dr. William V. Mullin, Cleveland: The matter of selecting the patient for either general or local anesthesia is very important, both from the standpoint of the patient and of the operator. I do not believe every case can be fitted to a local anesthetic, nor do I believe that every operator is fitted to do an operation under a local anesthetic.

Dr. Beck and Dr. Waugh both have asked me to say a word about their use of anesthesia. Dr. Beck does a great deal of work under synergistic anesthesia, while Dr. Waugh is rather partial to colonic ether. These men have used their methods in a great deal of sinus work, and very satisfactorily; but the point is this—something that Dr. Gallaher taught me—that if the frontal sinuses and ethmoid cells are extensively diseased and filled with hyperplastic tissue, the external operation is the only way to clean them out thoroly. It is results you want, and whether you use local or general anesthesia, you must get a thoro cleaning out of these cavities if you are to be successful. I do not refer to all types of cases but only to the chronic hyperplastic cases.

ALLERGY AS RELATED TO OTOLARYNGOLOGY.

W. W. Duke, Ph.D., M.D.

KANSAS CITY, MISSOURI.

Since the suggestion of Weichhardt and of Wolff-Eisner in 1905 and 1906, that hayfever seemed to be related to animal anaphylaxis, and since the suggestion of Meltzer in 1910, that bronchial asthma was a phenomenon of anaphylaxis, the subject of hypersensitiveness in humans as it affects the naso-respiratory tract, has grown to large and important proportions. There is marked similarity between animal anaphylaxis and a peculiar type of hypersensitiveness in humans. Whether or not the phenomena in animals and humans are identical, however, has not yet been conclusively proven. Suffice it to say, that they are remarkably alike in many respects. Most striking are the facts, that in each, an apparently harmless substance, such as an ordinary food stuff, can to a sensitive animal or human rank with the most violent poisons known to medical science, and that in each condition, almost every tissue and organ in the body is affected to a greater or less extent in a characteristic way. Few tissues are immune to the effects of reaction. Since surface tissues in humans come in contact with foreign agents more frequently than do the internal structures, it is surface tissues, such as mucous membrane of the nose and bronchial tubes, gastrointestinal tract and the skin, which are most frequently involved in reaction.

Nature of Agents Which Sensitize.

Apparently, an almost unlimited number of agents can sensitize a human and cause reaction. A serious effort has been made by many observers to determine if possible the nature of agents which tend to sensitize. Some little progress has been made in this line. Certain amines such as hystamin and tryamin, for example, can cause reaction in untreated animals or normal humans if given subcutaneously. The reaction is very much akin to that which follows a pollen injection in a pollen

sensitive individual. Drugs of the morphin series, likewise, have a tendency to cause a hive reaction if injected intracutaneously. Morphin, if given in dilute solution intracutaneously in normal individuals, causes a characteristic hive in practically every case. This is not true of other drugs, such as drugs of the cocain, atropin and quinin series. I tested intracutaneously a relatively large number of normal and allergic individuals with some twenty drugs and hystamin, and obtained positive reactions in relatively few except with hystamin and drugs of the morphin series. It appears that an additional factor, namely, mode of contact with foreign agents, is a very important factor, tho perhaps less so than the nature of agents which sensitize. For example, patients do not ordinarily react to substances which are met with frequently in gross amounts, such as the larger protein constituents of eggs, milk or the commonly eaten meats. They react rather to substances which are rarely met with, or substances which are met with commonly but in almost infinitesimal amounts. For example, several patients who reacted to beef extract proved to be sensitive to a constituent contained in beef in slight traces only, and which would stand boiling. They were not sensitive to the grosser proteins. Several cases who were sensitive to milk were apparently sensitive to none of the common constituents of milk, but to the article of food eaten by the mother or animal furnishing the milk. If a human were highly sensitive to the substances met with in gross amounts, the patient would either succumb or gain tolerance. Patients who are sensitive to pollen of course meet with only traces of pollen under normal conditions. They do not meet with enough pollen to give them tolerance. Our method of treatment, on the other hand, depends upon the systematic giving of pollen in ascending doses, until the patient gains tolerance and becomes able to handle the quantity which he meets with naturally. Slight surface contact with pollen is changed by treatment to gross intimate contact.

Types of Reaction.

An important distinction must be made between two types of reaction, namely, reaction in the nasal or bronchial mucous membrane, which is a result of direct contact between the membrane and some air carried substance, and reaction in

the same membranes occurring as a result of a constitutional reaction to some substance which has been absorbed and distributed by the general circulation. A food can cause reaction in either way. For example, egg eaten by mouth may either cause reaction thru emanations reaching the nasal passage during a process of mastication, or may cause reaction after partial digestion and absorption from the gastrointestinal tract.

Toxicity of Agents Which Sensitize.

Very few physicians except those who have had actual experience, can appreciate the high grade of sensitiveness which afflicts some individuals. To certain individuals, there is no poison known to medical science except diphtheria toxin and tetanus toxin which can act in such minute traces as a substance to which the individual is sensitive. The infinitesimal trace of the substance which can cause reaction can hardly be measured. For example, in a pollen sensitive case, I have known one ten-thousandth of a milligram of crude pollen to cause reaction. If the active substance in pollen responsible for reaction could be purified and used, I am sure that the quantity which could cause reaction would be reduced to one one-millionth of a milligram or less, that is one one-billionth of a gram. This should seem small to anyone. However, this fact can be illustrated possibly more practically by the citation of several illustrations. I recently observed two cases of egg dermatitis, for example, in children, who had been subject to eczema on the exposed parts for several years. They had been treated by several very capable physicians, and were sent to me with the correct diagnosis of egg dermatitis. In one case, the mother had realized that egg caused illness, and had carefully removed egg from the child's diet. In each case, several physicians had instructed the mothers to strictly keep the children out of contact with egg. On an egg free diet, under my care for several months, neither child showed improvement, and unsuccessful efforts were made to find some substance other than egg to which they might be reacting. In one case, I lost patience with the mother and told her I did not believe she was keeping the child on an egg free diet, and told her to keep eggs out of the house. She took me at my word, and returned later with a perfectly normal well child and a story that the dermatitis began to clear up from the day

she kept eggs out of the house. She found, that if either she or the father handled egg or ate egg while away from home, dermatitis would return when they handled the child. I immediately told the mother of the second child to follow the same procedure, with the result that the second child cleared up completely within a few days. Doctor Gerald B. Webb and Doctor Ray Matson have each cited to me an example of an egg sensitive case who could eat hen meat but could not eat rooster meat. I have been able to verify their observations in several cases. I have observed cases sensitive to honey, who would react with violent gastrointestinal attack after the digestion of one drop of honey. The patients, however, were not sensitive to honey of every type, but only to that derived from certain plants.

The Hereditary Constitution and Its Influence Upon Immunity.

Human hypersensitiveness is consistently a hereditary condition. One might almost say, that without heredity, human hypersensitiveness does not exist. This may be an exaggeration. A positive family history can be obtained in over half the cases of real pollen hayfever or asthma. If the families of hayfever cases are tested cutaneously, the percentage exceeds this. In other words, many patients have the tendency to hypersensitiveness but do not actually have clinical symptoms which attracts their attention. Individuals of allergic strain seem to belong, one might say, to a different race than that of nonallergic individuals. Their reactions are different. They are different not only in relation to reaction to material agents to which they may be sensitive, but also their reactions of immunity are different. Even their reactions to certain sensations, such as pain, heat or cold, are different in a fairly large percent of cases. Even their mental reactions to certain emotions are different in a rather large percent of the cases. For example, some individuals can hardly tolerate the pain caused by an intracutaneous injection of pollen, nor can others hardly tolerate pain caused by the heat of a nitrogen lamp, even tho the heat produced rarely exceeds that which the ordinary individual would call warm. Others can hardly tolerate the sense of cold on the skin. Others react to certain sensations of fear or worry, which would hardly concern the normal in-

dividual. The phenomenon of altered reactibility is by no means confined to such tissues as the nasorespiratory organs, but may involve the nerve cells as well.

I have found, that among the seasonal pollen cases, there is a high grade immunity to both tuberculosis and syphilis. Among perennial cases, we have a little different condition to deal with. These individuals, like the pollen cases, have a high grade immunity to tuberculosis and syphilis, but unlike the pollen cases, are more than normally susceptible to acute infections of the nasorespiratory tract, such as sinusitis and bronchial pneumonia. Among five hundred cases of hayfever, asthma and hives, which were carefully tabulated, only nine had active syphilis. (This is a much smaller percentage than is found among nonallergic individuals.) All responded well to therapy, and not one single case had evidence of tabes dorsalis or paresis. In fact, in my entire experience with allergy cases on whom complete physical examinations and Wassermann tests were made as a routine, tabes dorsalis of mild grade was found in only one case.

Among the same five hundred cases, in whom lung examinations were made carefully as a routine, only seven proved to have active tuberculosis. In an additional three, tuberculosis was suspected but not proven. Of the seven cases who had active tuberculosis, all were cases of long standing and rarely confined to bed. Only one case of tuberculosis observed during a period of ten years terminated fatally, and this was one of a cardiac death as a result of embarrassed pulmonary circulation.

The Relationship Between Chronic Reaction, Polyps and Sinusitis.

Other conditions commonly found among the perennial cases which were not characteristic of the seasonal cases were achylia gastrica, obesity, hypertension and hypotension. Of particular interest, also, among the perennial cases, is the fact that 24% of five hundred cases had nasal polyps, and 27% definite, unmistakable evidence of chronic nasal sinus infection. This ratio is quite different from that found among the seasonal cases, in which polyps were demonstrated in only 10% of cases, and chronic nasal sinus infection in only 6%. Inasmuch as deviated septum was found in about one-third of each

of these series of cases, this difference in statistics could not be accounted for thru this agency. It is believed that chronic edema of the membranes of the nose, continued thruout the year for several years, can lead to the formation of polyps, and that the mechanical obstruction caused thereby can predispose to acute or chronic infections in the nasal sinuses or bronchial tubes. Chronic edema may account for the high percent of polyps and infection in perennial cases. The same cannot be said of seasonal cases, even tho edema of the membranes may be more marked. It is believed, that in seasonal cases, the membranes regain normalcy during the well period, even if the illness continues for many years.

Agents Which Sensitize and Cause Reaction.

The various elements which tend to sensitize and cause reaction have been freely discussed in medical literature, and need only be mentioned at this point. The more important are the pollens, emanations of animal or vegetable matter of many varieties, smoke, dust, foods, drugs, insects, therapeutic sera and physical agents, such as light, heat, cold, mechanical irritation, and in the case of heat sensitiveness, mental or physical activity. Of the physical agents, heat and cold are the more important so far as otolaryngology is concerned. Many cases of nasal reaction which do not respond to material agents, will be found, if tested, to respond to the effect of heat or cold, or to the combined effect of the two. The symptoms which appear seem exactly the same as those caused by sensitiveness to material agents, and strange to say, may be either perennial or seasonal. I feel inclined to discuss this phase of our subject too freely and, for this reason, dismiss it at this point.

Diagnosis.

The symptoms of reaction are no doubt familiar to all of you, and need not be dwelt upon. The same may be said concerning diagnosis, altho I believe the majority of physicians who have had only slight acquaintance with the subject overemphasize the importance and reliability of skin tests. Those who believe that cutaneous tests give anything more than a clue to a diagnosis, are destined to many disappointments and

many mistakes. A diagnosis can be made with definiteness only by finding that relief of symptoms follows removal of the patient from contact with the suspected agent, and that symptoms can be again elicited thru again bringing about natural contact between the patient and the suspect agent. Even this is not conclusive proof, and shows only a location of the agent. This is the best we can do at the present time.

Treatment.

Treatment, of course, is of primary interest to all of us. Avoidance of the cause is, of course, the best and usually the simplest when this can be accomplished. If the cause cannot be avoided, specific treatment with the agent primarily responsible for the illness can be highly recommended. In the case of contact reaction between a surface membrane and something coming from the outside (as in pollen disease), the result is almost always good, if not perfect. Failure to get a good result should make one suspect either an error in diagnosis or a multiple cause for the illness. I have found this to be a fact with almost every case treated within a period of two years.

The routine fifteen dose methods given at specified intervals, recommended by some of the commercial houses, deserve to be condemned in strong terms as inaffective in a majority of cases, and actually dangerous. The response to pollen is not the same in all individuals, so that the rate of increase in dosage and the time interval has to be varied according to degree and time of response, also the ultimate dose has to be varied, not only according to the reaction of the patient, but according to the pollen content of the air in the district in which he resides. Even this varies in different seasons. Specific treatment with foods is not nearly so successful as treatment with pollen. This is probably due to the fact, that we are not always able to find and use the actual substance in the food which is responsible for the illness. Nonspecific and vaccin treatment has not seemed of much service in my experience, altho in rare instances a temporary good result can be obtained. Symptomatic treatment with adrenalin, ephedrin, atropin, acetyl salicilat is useful for temporary relief. I am not sure but that prolonged use of adrenalin several times daily, over a period of years, is undesirable. It seems to so

accustom a patient to its use, that it is difficult to relieve the patient by other means if adrenalin is discontinued. Habit forming drugs and anesthetics are contraindicated except in case of the gravest emergency. The morphin habit is much more easily acquired by allergy patients than by the average individual, and once acquired, is more difficult to relieve.

Nasal Surgery in Perennial Cases.

A word should be said concerning surgery in the case of nasal allergy or bronchial asthma. The allergic individual is different from the normal, and has to be handled differently both medically and surgically. This does not apply particularly to the removal of tonsils, and possibly only to a small extent to careful septal operations, when indicated. Even in the latter case, however, I have observed patients with perennial nasal allergy to be thrown into a state of chronic bronchial asthma after a septal operation, which was done with perfect technic and which was strongly indicated. It is possible that cases of this sort may be explained thru some variation in the cooling or drying of the membranes of the bronchial tubes following a widening of the nasal passages. Surely, it is difficult to account for otherwise. Operations on the turbinates and sinuses should be done with distinct fear in an allergy case. This may not apply to seasonal cases, but most certainly, does apply in perennial cases. Perennial cases have a natural tendency toward edema of the membranes and polyp formation. The removal of polyps does not remove the cause of trouble, and operation on the sinuses rarely relieves the condition except for the time being. Frequently, it leads to more and more operations and more polyps, and often to chronic infection. Patients with allergic noses have not the tendency toward perfect healing and recovery of normal membrane structure that is possessed by the normal individual, and the result is very often harm instead of good.

The Use of Sera in Hayfever and Asthma Families.

Finally a word should be said concerning the use of sera in hayfever and asthma cases. The otolaryngologist is no doubt frequently called upon either to use sera or advise its use in the case of diphtheria. Patients with hayfever and

asthma have a natural tendency to react to substances given intravenously, whether the substance be salvarsan, transfusion of blood or serum. Lamson, in an exhaustive review of the literature, reports the finding of a history of hayfever or asthma in 34% of the cases of death reported following the use of antitoxin. If a careful inquiry into a personal and family history of hayfever and asthma had been made in each of the cases reviewed, I think we could rely on the fact that this percent would have been grossly higher. It is hardly justifiable to give a prophylactic dose of antitoxin to a hayfever, asthma or hive case in the case of exposure to diphtheria. It is better to allow the patient to chance contracting the disease. Furthermore, it is hardly justifiable to give a prophylactic dose of tetanus antitoxin unless very strongly indicated. The disease, diphtheria, of course, almost necessitates the use of antitoxin. When given therapeutically, however, it should be given according to a special technic recommended by Crea and the dose administered should be reduced to the minimum.

DISCUSSION.

DR. WILLIAM V. MULLIN, Cleveland: Dr. Duke is an optimist, and I think that particular type of man is needed to treat these allergy cases. It must be a tremendous satisfaction to say that the treatment is almost universally successful, and that he can help 100 per cent. of cases of hayfever, etc. But I think the whole subject ought to be approached from another end. Start with the failures and find out why. Do not be overencouraged by some success. If you are successful be thankful, but do not think all your cases are going to come out that way. It seems to me, that if some of the time that has been put on trying to immunize people and trying to find out their reactions were put on endeavoring to find out why they became sensitive in the first place, we might get somewhere. Dr. Duke admits that he does not know why they become sensitive. But that is where we have to start in this investigation. I do not believe they become sensitive to just one offender—they will be sensitive to other things. That has been proven in the hayfever problem. People living in various localities will be sensitive to their environmental pollens, and if they move away from the Rocky Mountain district to the Pacific Coast or to the Middle West, they may be temporarily free from hayfever, and then they may become sensitive to what they are exposed to there. And very often you will get a case thoroly immunized to a certain pollen one season, and next year you will find him sensitive to some other pollen.

As to the tuberculous being less sensitive to allergy, I do not agree with that either. There is a sanitarium in this vicinity that has somewhere around 250 patients, and they admit only tuberculous individuals. The location is out where they are exposed to the wind, and therefore

the wind borne pollens, and a great many of these patients have both hay-fever and asthma.

I think asthma cases should be classified. Some are due to allergy and irritants, while others are due to infection, particularly in the nasal accessory sinuses of the hypertrophic type.

Dr. Duke says that allergy causes polyps, but on the other hand, I believe firmly that infection in the nasal sinuses is also a cause of hypertrophic tissue and polyps, and that given a case of bronchial asthma with well developed hyperplastic sinusitis, whatever the cause, with the nasal cavities full of polyps, you will not help him so long as you leave those sinuses full of polyps.

Dr. Ralph A. Fenton, Portand, Ore.: I think we should be grateful that so thoro and enthusiastic an investigator as Dr. Duke has presented this subject, even tho we may disagree in regard to certain individual questions as to technic, and so forth. I think we should also feel, perhaps, that there might be some searching of hearts among otolaryngologists, because the average specialist has been encouraged to undertake the investigation of allergy on his own hook. We have had proprietary gentlemen come around with enticing literature, and they have encouraged us, without special knowledge either of the exact pollens or the exact dangers of those pollens, to undertake certain investigations in which some of us have gotten into more or less grievous trouble. I think perhaps this should point a warning that in most cases, when it is reasonably possible, these cases should be turned over to some expert allergist. There is a particular reason for this in the fact which I think Dr. Mullin alluded to —that the pollens are essentially local, especially in the seasonal types, and these tests are essentially autogenous affairs, which should be studied for the individual case. For example, I had a case of a woman who recovered when she came across the Cascade Mountains to the Pacific side. She had been under treatment for asthma at Reno and Salt Lake City and numerous other intermountain localities, and she had never been tested by an allergist. When she was tested, our allergist found her to be sensitive to every sort of sage brush there was in the world! Of course, so long as she stays on the other side of the Cascades, in which country there is no sage brush, she remains immune.

As to the question of foods, how about the hypersensitive child? If he has all sorts of food taken away from him—eggs, milk, rice, spinach—what can be done for that child? He will starve if you take all those things away from him. I have in mind one food sensitive child to whom we gave small quantities of adrenalin, and it straightened out nicely the acute difficulties. That child now gets a small quantity of adrenalin by mouth, two or three drops on the tongue, and he is better immunized now than when we attempted to free him from the various foods.

I agree with Dr. Mullin that we should not perhaps accept in full the operative prohibitions imposed upon us by the allergist. We must get purulent discharges out of the nose and throat in some way. If these noses are left plugged up, especially in winter time, there is bacterial invasion with swelling, and when that occurs there is no drainage. We have recently adopted a rule (each case is a law unto itself) respecting leaving of erectile tissues alone in the nose. We snare out polyps and slip under turbinates without damaging the mucosa. In submucous resection, we

make as little scarring as possible. What we are doing is to get drainage with very little damage to the erectile tissue of the nose, which is so closely connected with the sympathetic system. We have found that these people are especially susceptible to the hemorrhagic type of organism, not only the streptococcus, but also the staphylococcus.

As to the danger of reaction in the administration of allergins, the history must be carefully taken in all such cases. We started to administer a series of allergins (grass type) in one woman, who gave a tremendous reaction the first week. The material was prepared by a competent allergist in our community, but on the second test she gave a more violent reaction than the first. Then we found to our sorrow that she had been taking thyroid tablets to reduce her weight. Our allergin reaction had started trouble with the thyroid gland, being just enough to initiate a toxic reaction from the tablets—a serious complication.

Dr. Harry P. Finck, Boston: I wish to thank the Academy for this opportunity to express my ideas and to give a preliminary report on the research work I have been doing on the nasal mucosa during the past six years under the guidance of Dr. Mosher of Boston. I have approached this particular subject from an entirely different point of view from Dr. Duke, but I am glad to say that my results from the microscopic and pathologic point of view are pointing in exactly the same direction as his.

The old opinion as expressed in the older text books was, that when there was polypoid or edematous swellings in the nasal mucosa, they were sometimes due to a vasomotor disturbance as well as to bacterial infection. This theory has been displaced by the infection theory of late years, that is, that chronic infections were the etiologic factors in causing practically all polyps in the nasal mucosa. In the past six or seven years of my observations, at least twenty percent of the polyp cases are due to vasomotor factors or vasomotor background. As I will point out later, this percentage may be greatly increased. The distribution of the vasomotor polyp occurs thruout the nose, not only in the nasal fossae, but also on the turbinates, in the nasal meati, and in all the sinuses. Sometimes these polyps are merely small edematous or polypoid areas which can hardly be seen, or they may be firm, large and pedunculated. Sometimes they are large and diffuse, and sometimes they are large enough to protrude out of the antrum down into the nasopharynx.

Briefly, there are two types of these polyps, acute and chronic. The acute type is a transitory type, which disappears after a severe vasomotor attack has left the patient; or they may appear only during the height of the attack. Microscopically, these transitory polyps are nothing more than an edema of the mucous membrane. This is probably due to the reaction of the vasomotor nerve mechanism controlling the cavernous vessels and lymphatics. The characteristic tissue cell that we find here is the eosinophil. The eosinophil is apparently the characteristic cell in allergic conditions. The chronic type of polyp is more permanent. As Dr. Duke pointed out, you can have a prolonged stimulation, a persistent vasomotor disturbance, and this edema causes these polyps to persist. Connective tissue is laid down and forms a firm substantial framework for the polyp. Besides eosinophiles, plasma cells then appear in the polyp stroma in varying numbers. The plasma cells usually mean a chronic inflammatory reaction by

the tissues to the ordinary bacterial exposures and attacks, or possibly also, to a slight extent, to mechanical exposure and trauma in the nose.

It is very important to recognize the fact, that when one has a prolonged or chronic vasomotor reaction in the nasal mucosa, superimposed pyogenic infections can occur. Often they do not. These secondary pyogenic infections can cause true purulent pus and other pyogenic tissue reactions in all parts of the nose. Thus, many cases of recurrent colds, sinusitis, pansinusitis, and chronic rhinitis have their underlying etiology very much obscured, and as a result these patients keep coming back for operation after operation, much to the surprise of the rhinologist. The obscure underlying vasomotor disturbance must be controlled before satisfactory relief or cure is obtained in this large group of cases.

The various types of polyps will be described and classified etiologically by their microscopic picture in some papers which I am preparing. We hope that this work will help materially in deciding on the etiology, treatment and prognosis of various types of nasal polyps and of pathologic nasal membranes.

Dr. E. N. Robertson, Concordia, Kans.: The idea that comes to me in this connection is operating on cases of vasomotor rhinitis. For a number of years, I have been in the habit of inquiring about the diet of people who come for operations on the nose, or for adenoids or tonsils, especially children, and I have found a number of cases that have been sensitive to ordinary foods without even giving them a single test, that is by telling them to eliminate a certain food for a week, and another food for the second week until we found the thing to which they were sensitive. Many times their symptoms disappeared and operation was not necessary, even in cases where the septum was deflected and it looked as tho that was the cause of the trouble. One case of special interest was a little boy who came in with a typical vasomotor rhinitis. The history was, then when he was two years old, he had a blocking of the nose, and since then many head colds and continued difficulty in breathing thru his nose. He had had his adenoids and tonsils removed and all indicated treatment, with the symptoms unrelieved. Three years later, he had a second operation, but still the symptoms continued. We found he was sensitive to eggs and told him to cut out eggs, not even to eat any chicken. He had a return of the trouble after eating chicken gravy, and even ice cream, but since discontinuing all foods with even a trace of egg or chicken, there has been no return of his symptoms.

Dr. W. W. Duke, Kansas City, Mo. (closing): I wish to express my sincere thanks for this kind discussion. I am afraid I did not make myself altogether clear on all the points, for example, in regard to obtaining 100 percent relief in all pollen cases. I do not mean to say that pollen treatment is ideal in every pollen case, for in some we find sensitiveness to more than one agent. In this case, pollen treatment is only partly successful. The cause of an unsuccessful result is usually multiple sensitiveness. A rather common additional finding in pollen cases is sensitiveness to heat or cold. In this case, symptoms may recur in a successfully treated pollen case toward the end of the fall season, when the weather becomes cold and changeable.

Concerning Dr. Fenton's question regarding multiple sensitization, a great many people are sensitive to more than one agent. However, they

are rarely sensitive clinically to all the agents to which they give positive skin tests. For example, a person may be sensitive to egg cutaneously, but the gastrointestinal membrane may not absorb the exact chemical body in egg to which the patient reacts. In thise case, he may not be clinically sensitive. As a rule, children are not sensitive to a great variety of agents. More often they react to something commonly found in many places, and give the impression of multiple sensitiveness.

As to polyps, I do not wish to minimize the part played by infection in the pathogenesis of polyps. I realize fully that infection causes polyps. I do also believe that chronic edema of the nasal membranes predisposes to infection, and that such infection can cause polyps. I believe in addition, however, that there is a type of polyp that comes primarily from allergy itself, especially in the perennial type. An allergic mucous membrane is not a healthy membrane, and does not always return to normal after prolonged continuous reaction.

Without saying too much about the treatment of polyps, I must say I have seen polyps grow smaller under the influence of both the specific and symptomatic treatment of allergy.

Dr. Finck's study is very interesting and valuable. He shows definitely that allergy can cause polyps by finding eosinophiles in them. The eosinophil is the characteristic cell of local allergic reaction. The characteristic cells of inflammatory reactions are polymorphonuclear neutrophiles, lymphocytes and plasma cells. In finding polyps infiltrated with eosinophiles, I think he proves the point which I have been so anxious to bring out.

FAULTY NUTRITION AS A CAUSITIVE FACTOR IN INFECTIONS OF THE UPPER RESPIRATORY TRACT.*

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It has been observed, that rats fed diets adequate in all other respects, but low in the Vitamin A factor, in the course of time develop infections of the upper respiratory tract. Such animals manifest many of the symptoms of human beings suffering from similar types of infections—for example, loss of appetite, failure to gain (in children), gastrointestinal disturbances, snuffles, nasal catarrh and death, unless appropriate treatment is introduced. Macroscropic examination of the various organs of such animals, with the exception of the nasal passages (sinuses), which have been found filled with pus, shows nothing consistently abnormal. Occasionally, the lungs are infected; often the intestines are filled with gas and greatly distended. Death in these animals seems to be due to an infection superimposed on tissue which have been altered by the dietary deficiency. In those cases where the animals have been able to continue for longer periods of time on the restricted rations, the peculiar eye changes characteristic of xerophthalmia have developed.

The similarity between the infection of the upper respiratory tract in man and in our experimental animals is emphasized by a comparison of the pathogenic organisms present in the two species. Smears from the nasal passages of those animals presenting the characteristic syndrome of the respiratory infection consist of staphylococcus albus and staphylococcus aureus. In some cases the hemolytic streptococcus was found.[1] These same organisms are invariably present in the smears of the nasal passages of children suffering from sinus and mastoid infections, and seem to be the cause of the untoward symptoms. It should be noted, how-

*From the Child Welfare Research Station, State University of Iowa.

ever, that these are found in smears taken from apparently normal rats and well children. In rats, the untoward symptoms follow only in those animals which have been fed diets low in Vitamin A.

Investigations by Blegvad[2] on the incidence of xerophthalmia in Denmark, have shown that the disease is inversely proportional to the consumption of butter fat (milk, butter and cheese), which is the chief source of Vitamin A in the diets of the Nordic races. During the earlier years of the War, when dairy products were being more largely exported, there was a considerable increase in the number of cases of xerophthalmia. For example, in 1909, when there was a daily average consumption of 40 grams of butter per capita, 20 cases of xerophthalmia were reported; in 1926, with an average consumption of 15.3 grams of butter fat per capita, 78 cases were reported; whereas, in 1917-18-19, when the exporting of butter was forbidden and there was an increased home consumption (41 and 51.5 grams respectively per capita), xerophthalmia almost completely disappeared. In Sweden, where the daily per capita consumption of butter fat amongst the poorer families averages 53 grams, xerophthalmia is unknown.

In the United States, very few cases of xerophthalmia have been reported. However, many of the symptoms associated with the condition are not infrequent. In the 165 cases collected by Blegvad, the following complications were recorded: Pneumonia, 63; bronchitis, 45; pyuria, 42; otitis media, 30; nasal catarrh, 26; subcutaneous abscesses, 4. The slight resistance of these patients to infection was particularly noticeable.

Since a low Vitamin A consumption in animals, and in the inhabitants of Denmark, seems to be associated with infections of the upper respiratory tract, an attempt was made to see if there is any correlation between the butter fat consumption and the prevalence of sinus infection in certain sections of the United States. Data (Table I) obtained from the United States Dairy Statistics show, that there is considerable variation in the amount of butter consumed in different localities, the lowest per capita average being 19.7 grams in Lewiston, Idaho, and the highest, 44.6 grams in Missoula, Montana.

Since these figures are averages, it is obvious that some individuals are taking more and others less. The following table (Table II) shows great variation in the per capita con-

sumption of butter fat in a number of economically safe family groups. Most of these are college faculty families.

It will be noted that the average consumption is less in some cases than that which is considered safe in Denmark, and considerably less than that reported as being the average of the poorer families in Sweden (53 grams per capita). Foods other than milk fat contain certain amounts of Vitamin A (Table III). These are being used in varying amounts, and may explain why xerophthalmia is not more frequent in the United States. However, even when these are being used, it is obvious that the Vitamin A content of the diet is dangerously low in certain instances, since the consumption of other Vitamin A containing foods is relatively low in most dietaries. This low Vitamin A content of the average diet, we believe, is responsible, in part at least, for the low resistance of individuals to various types of upper respiratory infections.

The high incidence of upper respiratory infection in the artificially fed infant in comparison with the low incidence in the breast fed babe, has until recently caused some doubt concerning the validity of our thesis, for a baby on a well constructed cow's milk formula would seem to be getting enough butter fat and, therefore, enough Vitamin A. The explanation for the conspicuously greater immunity in the breast fed baby, however, seems to lie in the better absorption of the Vitamin A in the breast milk. Rats which were suffering from the infection which follows the Vitamin A deficiency, were given rations containing as the only source of Vitamin A the ether extract of the stools from an artificially fed baby. These animals immediately began to gain in weight, and quite recovered from the effects of the infection. Another group of Vitamin A depleted animals were given the ether extract of the stools of a breast fed baby, who had been receiving in addition to breast fed milk orange juice and 1 teaspoon of cod liver oil daily. All the animals in this group died, thus demonstrating the greater availability of the Vitamin A in the breast milk. The Vitamin A in the artificially fed baby is apparently very largely occluded in the calcium soap formed during the digestion of the cow's milk.

It seems obvious that an increase in the consumption of those foods which are rich in Vitamin A will protect individuals from such infection of the upper respiratory tract. However, once the infection has become firmly established,

operative intervention may be necessary, altho in certain cases, right dietary treatment may be all that is needed to correct the condition.

In testing out the dietary therapy, it is necessary to include not only foods rich in Vitamin A, for example cod liver oil, green leaf vegetables, milk and dairy products, but the other essential food constituents must be included—protein enough to cover the body needs, inorganic salts, especially calcium, phosphorus, and iron.

Foods rich in Vitamin B (succulent vegetables and fruits), are also essential, since they aid in stimulating the appetite, which is often inadequate in these patients. Vitamin B also is of assistance in bringing about normal peristalsis. The carbohydrate needs of the body may be supplied by the more readily absorbable sugars in the fruits and succulent vegetables, prepared cereals and well toasted whole grain breads. Since the cereal foods supply practically no Vitamin A and little Vitamin B, these are given only after adequate amounts of the other more essential foods are taken.

Patients suffering from sinus infection suffer from digestive disturbance, manifested by low gastric secretion, vague pains, and gas. To overcome these difficulties, we give foods in a finely divided form. That is, nothing is served which will enter the stomach or remain in the stomach in chunks, and no nonsterile food is given, the only raw food allowed being that which comes put up by Nature in sterile packages—for example, citrus fruits and ripe bananas. All meat should be well cooked and as free from fat as possible. Any pastry served, such as cake, pies or cookies, should be made as rich as possible with butter and eggs, since by this means we are including Vitamin A. All foods served should be in such palatable and digestible form, that a sufficient amount will be taken to meet the needs of the individual.

TABLE I.

AVERAGE DAILY PER CAPITA CONSUMPTION OF MILK AND BUTTER FAT IN SOME TYPICAL CITIES.

City	Total Milk Equivalent pts.	Total Milk Butter Fat gms.	Total Butter Fat gms.
	CALIFORNIA.		
Napa	1.285	20.2	37.2
San Francisco	0.709	10.6	27.6
Oakland	0.963	14.4	31.4
	FLORIDA.		
Sanford	0.251	3.7	20.7
Miami	0.394	5.9	22.9
	IDAHO.		
Lewiston	0.187	2.7	19.7
Boise City	1.561	23.4	40.4
	IOWA.		
Pella	0.247	3.4	21.2
Colfax	0.750	11.2	29.0
Boone	1.000	15.0	32.8
Eldora	0.305	4.5	22.3
Cedar Rapids	0.767	11.5	29.3
	KENTUCKY.		
Henderson	0.205	3.0	20.0
Danville	0.650	9.7	26.7
Winchester	0.310	4.6	21.6
	KANSAS.		
Parsons	0.635	9.5	26.5
Dodge	0.234	3.5	20.5
	LOUISIANA.		
Shreveport	0.626	9.3	26.3
	MISSOURI.		
St. Louis	0.654	9.8	26.8
Chaffee	0.388	5.8	22.8
	MASSACHUSETTS.		
Cambridge	1.435	21.5	39.3
Boston	1.120	16.8	34.6
Fall River	0.594	8.9	26.7
Framingham	0.721	10.8	28.6
Glouchester	0.987	14.8	32.6
Provincetown	0.235	3.5	21.3

Montana.			
Missoula	1.842	27.6	44.6
Lewistown	1.459	21.8	38.8
North Dakota.			
Manden	0.637	9.5	26.5
Valley City	1.080	16.2	33.2
New York.			
New York City	1.160	17.4	35.2
Buffalo	0.697	10.4	28.2
Rhode Island.			
Newport	0.882	13.2	30.2
Providence	0.927	13.9	30.9
South Dakota.			
Sioux Falls	1.417	21.2	38.2

Average butter consumption for the United States was 17 pounds per year, or 17.8 grams butter fat per person per day.

Table II.

Daily Per Capita Consumption of Milk and Butter Fat in Economically Safe Family Groups.

Family	Milk Equivalent pts.	Total Milk Butter Fat gms.	Butter gms.	Total Butter Fat gms.
F-F	1	16	28	36.3
H-B	1.78	27.1	56.5	78.9
G-B	2	32	56	86
T-B	1.66	34	43.1	70.3
M. D.	2	32	47	71.9
T. D.	1	20	31	46.3
T. Z.	2.5	37.5	64.2	92
P. H.	1.09	17.5	16.	31 1

TABLE III.

DISTRIBUTION OF VITAMINES IN COMMON FOODSTUFFS.

	A	B	C	D
MEAT, FISH, ETC.				
Lean meat (beef, mutton, etc.)	+	+	+	
Liver	+	+	+	+
Kidney and brains	++	++		
Fish	+			
DAIRY PRODUCTS.				
Butter and cream	+++	0	0	+
Milk (whole, raw)	++	++	++	+
Milk, (whole, pasteurized or boiled)	++	++	+	+
Egg yolk	++	++	0	++
VEGETABLES.				
Cabbage (raw)	++	++	++++	
Carrots, parsnips, spinach, beets	++	++		
Lettuce and tomatoes (raw)	++	++	++++	
Potatos and onions	0	++	+	
Peas, beans (dried, cooked)	+	++	?	
CEREALS.				
Wheat, corn, rice, oats, barley (whole grain)	+	+++	0	
White wheat flour, polished rice	0	0	0	
Wheat, rice (germ)	+	++++		
Wheat (bran)	0	+	0	
Germinated grains and cereals	+	++	++	
FRUITS.				
Oranges, lemons, grapefruit		+++	+++	
Grapes, grape juice, raisins	0	+	+	
Pears, apples, limes	0	++	++	

Fats and Oils.				
Cod liver oil.....	++++	0	0	
Lard, corn oil, olive oil and vegetable oleomargarin	0	0	0	0
Mutton and beef fat....	+			
Miscellaneous.				
Yeast	0	++++	0	
Malt extract, meat extracts	0	+	0	

0 None. + Very little. ++ Some. +++ Considerable ++++ Very rich.

Vitamin C prevents scurvy.

Vitamin B important in controlling appetite and normal peristalis.

Vitamin A decreases incidence of respiratory infections; prevents ophthalmia and night blindness.

Vitamin D not a vitamin, probably, but radiant energy. Prevents rickets.

Adapted from the "Vitamins"—Sherman and Smith.

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1. For the bacteriologic studies I am indebted to Mr. L. J. Belding, Bacteriologist, Pediatric Department, University Hospital, State University of Iowa.
2. Blegvad. Quoted by E. Wedmark, Lancet, 1924, vol. CCVI, p. 1206.

DISCUSSION.

Dr. J. A. Stucky, Lexington, Ky.: I have repeatedly verified, in the human child, the truth of the statements made by the essayist as to faulty nutrition being the cause of infection in the upper respiratory tract, and it is a source of great satisfaction to know that these facts have been demonstrated in the laboratory on lower animals. I still insist that there is a difference in the metabolism of the human being and that of the guinea pig, rat and rabbit.

For the past two or three years, while I have been excluding refined sugars, highly milled cereals, flour and meal, because of the repugnance of many of the children to eating cereals without sweetening of some kind, I have used either malt sugar, honey or old fashioned brown sugar, unrefined, or fruit juices and jellies, and have had no evidence of toxemia as produced by ordinary cane sugars. I have also gotten most satisfactory results on the minimum balanced diet suggested by the essayist, which was mentioned in my paper before the American Laryngological, Rhinological and Otological Society in St. Louis, May, 1924. From a clinical standpoint, I am convinced that our susceptibility to bacterial infection, and the reason we are finding so many bacteria, cocci, etc., in the ears, nose and throats of our young people is because of the lowered power of resistance caused by the denatured and devitaminized food we eat.

I congratulate the Academy that so reliable an authority calls our attention to the possible solution of the greatest problem confronting the medical profession, especially the otolaryngologists, the problem of nutrition. Not only is the mucous membrane lining of the accessory sinuses made to suffer as a result of the infection, but we are raising a nation whose bones, especially the teeth, are deficient in calcium, and there is an increasing number of proven cases of endocrin imbalance, all showing the urgent necessity of remineralization, which can only be satisfactorily done by means and methods given us by Nature. If this is empirical, then Nature is empirical, but this remineralization cannot be obtained from the pharmacist as satisfactorily as it can be in Nature's food products.

Dr. John F. Barnhill, Indianapolis: I have been greatly interested in this presentation of Dr. Daniels. I believe it is timely, and that we will in the future treat our patients better for having given them the thought she has passed on to us.

There is one point which I understood Dr. Daniels to make that I feel might well be challenged. I got the impression from her discussion that she believes infection of the sinuses is caused by a lack of feeding Vitamin A. In other words, if an individual has sinusitis, the disease results from lack of this vitamin in the food. Dr. Daniels probably does not mean to give this impression.

Possibly newspaper men are present, and if so they may herald an entirely erroneous statement to the public as to what the essential cause of infection and inflammation actually is. Personally, I do not understand that infection ever is possible except from one cause, namely, the presence and activity of numerous pathogenic bacteria.

I take it, therefore, that the lack of Vitamin A is a predisposing cause of very great power, but not the real cause of the infection, just as the presence of an adenoid is not the real cause but a predisposing cause so potent that the child having the adenoids will more readily take on sinus infection, especially if he does not get his Vitamin A. I should like to hear Dr. Daniels' opinion as to whether or not she thinks the lack of Vitamin A will, without other causes being present, actually cause inflammation and infection.

Dr. Oliver P. McCartney, Kansas City, Kans.: This is a subject which has been of interest to me for many years; in fact, before I quit general practice. I had the pleasure of practicing medicine out on the western slope of this state when the Gunnison Tunnel was being put thru the mountain to carry the water of that river into the Incompahgre basin. You will remember, when the tunnel was part way thru, the money gave out and it was not finished until about seven years later. The result was that the homesteaders went in there and lived in little one room tar roofed cabins, with insufficient food, poor water, and all the hardships which go with homesteading in arid regions, altho the milk supply was better as a rule than is found in the populous centers, but little of it was to be had by these people.

Colorado is notoriously free from tubercular trouble in its native population, so this could be ruled out as a predisposing cause. It used to be said that a native could not acquire tuberculosis in this climate, but that fallacy has long been exploded. I have had babies under my own care

who would get an attack of indigestion with all its train of symptoms; they would become emaciated, and in spite of anything that could be done in the way of modification of milk and the varying of such other food as was to be had, the condition would get steadily worse. I have seen the middle ear break, the corneas slough, and the child die within three days, where the food was overwhelmingly carbohydrate in content and deficient in fat soluble A. I have seen the tissues break down in these marasmic children, the knees become boggy and fluctuate, and if the child had vitality enough to keep it going, there would be a discharge of pus looking substance, in the course of time, later to return to perfect function if the necessary vitamins were fed and assimilated, showing the pathology was produced from nothing but starvation. Dr. Daniels stressed the cheapening of foods by omitting butter, eggs, etc. Had time permitted, she might have told of the one minute beverage, the two minute dessert, the three minute breakfast food, etc., to say nothing of the antebellum canned foods and cold storage supplies which have lost everything but the flavor which naturally belongs to food. The same havoc is wrought in most dehydrated vegetables so much used in making restaurant soups. The Vitamin A normally comes from the leaves of various vegetation, and is not in the milk of cows not fed upon such.

Strangely enough, the cod fish, tho not a vegetarian and living beneath the water almost wholly, has a large supply of this precious material in its liver, most probably due to its large consumption of squid which produces the Vitamin A, possibly to compensate for its sunless surroundings and the inky secretion with which it intensifies the natural subterranean fog. Cod liver oil carries Vitamin A and D, yeast a bountiful supply of B, and the citrus fruits C. Tomatoes contain Vitamin A, B, and C, and are injured very little in cooking, altho all suffer with prolonged heat, especially if such heat is in the presence of oxygen and alkali. In the low lying countries, where the benefit of continuous sunshine with its short wave lengths is not to be had, the problem is a little more difficult. A special light may be provided to generate these rays, but we must bear in mind that too much actinic ray is about as bad as too little.

Dr. A. A. Hayden, Chicago: Dr. Daniels refers to a visit I paid to her clinic last week. I want to tell you something of the impression that I got in the few hours I spent there. Her work, as you know, is this: The University of Iowa, in connection with the Child Welfare work there, and especially in the baby wards, have a considerable number of children with manifestly diseased sinuses appear for treatment. The bacteriologic count in those children with nose and throat disease does not differ materially from the children that are not so diseased. This comparative bacteriologic examination permitted these people to seek the cause of why in one case the sinus condition developed, and why in another, under the same circumstances apparently, nothing happened. That led to experimentation with rats which were handy animals to experiment with, and the doctor can actually, as I saw, run these rats up and down the scale into sinus condition or out of sinus condition, by increasing or diminishing the amount of Vitamin A in their food.

One of the very best cases—and I had these rats brought in here so Dr. Daniels can show them to you—one of the very best cases which Dr.

Daniels had and which she tried to preserve alive for this meeting, unfortunately died. There is, however, in one of these cages a rat similarly affected, but whether it will sneeze for us while Dr. Daniels is exhibiting it, I do not know, but while she was reading her paper it manifested all the symptoms, as far as sneezing is concerned, of an acute head cold.

Another thing I want to note in passing is that, as you understand, we have a family of rats in one of these cages, and most of the time this afternoon this family has been at a dinner of carefully prepared food. These rats, when I was in Iowa City, four or five days ago, were about the size of the end of this lead pencil; they are very much larger than that now, being almost as large as my thumb.

I think Dr. Daniels' paper has demonstrated to us the truth of the statement of Napoleon, that an army really fights on its stomach, and that we really dig our graves or prolong our live by the food that we eat or do not eat.

Dr. Amy L. Daniels, Iowa City, Ia., (closing): When we first observed that the rats on the low Vitamin A diets were prone to infections of the upper respiratory tract, we isolated them, fearing that the infection might be passed on to our other experimental animals. As the number of animals in our experimental groups increased, it was not possible always to carry out isolation technic, and animals receiving good rations were often placed in cages adjacent to the infected animals. To our surprise, we found that the well nourished animals, that is, those receiving diets high in Vitamin A, never seemed to get the infection. Since we had been working on the theory that the Vitamin A low animals were suffering from a peculiar type of infection, our findings were a bit disturbing. However, recent work seems to corroborate our results. Several weeks ago, our pathologist, who had become interested in sinus infections, attempted to produce the condition in dogs, which up to this time had been well fed. Organisms isolated from the sinuses of rats, and children suffering from infections of the upper respiratory tract were injected into their nasal cavities. All results were negative. The dogs failed to show any of the symptoms of the upper respiratory infections. Normal tissues apparently are not affected by those organisms or their toxins which produce such diastrous results in tissues low in Vitamin A.

HEADACHES—THE NASAL ASPECT.

Ross Hall Skillern, M.D.

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The interpretation of headache resulting from inflammatory conditions within the mucosa of the accessory sinuses, has undergone rather curious phases during the past few decades. In the beginning, it is needless to say, the true significance of these pains was naturally but little understood by the general practitioner or, for that matter, by the specialist either, and many of these patients underwent wide and varied cures, such as climatic, dietetic and even hydro- and electrotherapeutic, without, of course, the slightest change in the ultimate trend of the disease, and little in the symptoms referable to the head.

Then came the period when any headache from a sinus infection was described as typical, that is, always being situated over a certain small area, depending upon the sinus involved, and most significant of all, appearing at a certain time in the morning and disappearing in the afternoon with equal rapidity. If the head pain did not conform to these, it could not have originated from an inflammatory sinusitis.

Now, the latter day and modern aspect has so changed and augmented these views, that it has, indeed, become a study in itself to draw reliable deductions from head pains concomitant with a case of chronic multiple sinusitis. To make this clear, let us assume that we have a patient suffering with a frontal and maxillary sinusitis of equal severity on the same side. The symptoms should consist of a frontal pain from the frontal inflammation, and a feeling of fullness in the malar region, with disagreeable sensations in the alveolar process and probably the teeth, from the maxillary involvement. As a matter of fact, all of the symptoms may be confined to the supraorbital region, thus entirely obscuring the maxillary infection, leading one to believe that the trouble was limited to the frontal sinus. Unfortunately, when there is a combined infection of two or more sinuses, the head symptoms do not necessarily take on a combined form of like intensity, but one

may so overshadow the other, as to completely deceive so far as the actual sinus affected is concerned, to say nothing of the extent or degree of pathologic involvement present.

We will, then, see that the phenomenon described as headache, resulting from pathologic conditions within the sinuses, varies so greatly as to constitute perhaps one of the most inconsistent and variable evidences in the domain of symptomatology; happily, however, diagnostic clues of much importance may be gathered from this apparent confusion, for, as the old adage has it, "The exception proves the rule," so here the very inconstancy of the headache must bring to mind, certainly to the rhinologist and perhaps also to the astute internist, the probability of a nasal sinus involvement.

Preliminary to discussion of the individual sinuses with the special characteristics attributed to them, it might be well for a moment to consider the actual pathologic factor which gives rise to the subjective sensation of pain.

Headache, both as to character, degree and even time of appearance, depends largely upon the pathologic condition present within the sinus, the most important being some form of pressure. This pressure may result in two ways. One, in which the lining mucosa of the sinus becomes so congested and swollen as to impinge on the lining mucosa of the opposite side, thus meeting and obliterating the sinus cavity, or at least converting it into a slit like aperture. The other results when, from one cause or another, the drainage passages become more or less occluded, thus causing partial or complete stagnation within the sinus, with resulting pressure on the already swollen and inflamed mucosa. A third form which I mention, not so much on account of its importance, as for the sake of completeness, is the negative pressure or socalled vacuum headache. While this may play a certain role, nevertheless, I believe its relative appearance as well as importance have been exaggerated, for as my experience advances, it would appear that this type of headache is becoming less and less in evidence, and certainly more and more difficult to prove to one's entire satisfaction.

The pain, then, is due to pressure exerted upon the nerve endings in the sinus mucous membrane, associated probably with a certain toxicity liberated by the infecting microorganism. We would, then, have both a mechanical and a toxic factor in the causation of this subjective phenomenon known as headache.

Accepting this as true, what then are its characteristics? Do we have a typical sinus head pain? Broadly speaking, as already mentioned, this can be answered in the affirmative, but with reservations. With the ordinary text book case of sinus disease, running the usual course, on questioning the patient regarding his headache, he would state that the pain comes on shortly after arising and continues until 1 or 2 o'clock, then suddenly ceases, and he is comfortable for the balance of the day. That, as we have seen, is the typical sinus headache, but where one is typical, a dozen will be quite the opposite, so that unless it happens to be one of the variety adhering strictly to type, so far as periodicity is concerned, little reliance can be placed upon its diagnostic import.

What, then, regarding the character of the pain, referring especially to its degree and type?

The character of the pain may be dull, splitting, semithrobbing, throbbing, and combined, the old sick headache of our boyhood days. All these depend upon the pathologic process or condition present in the sinus or sinuses at that particular moment. The presence of any one of these types has its own peculiar reason, and one can accept with reasonable certainty that the severity of the pain is directly as the congestion in the sinus and sinus mucosa. Thus, if the sickening, throbbing symptoms prevailed, one can rightly deduce that an acute period of passive congestion, or rather an active congestion, is at the moment in the process of being, while should only a dull ache be present, either the acute process has subsided and is in a state of comparative quiescence, or the infection is a mild one and has not greatly affected the sinus mucosa. Even this statement, however, cannot be accepted at its apparent face value, but has its limitations, as will be seen when we speak on the frontal sinus.

From the foregoing, it will be seen that in sinuology, one endeavors to associate a given type of pain with a recognized pathologic condition within the sinus, and one of the results of this is that certain obscure types of cephalalgia are no longer being recognized; chronic nervous headache, for example, is a term which is now rapidly becoming obsolete.

Another characteristic of the sinus headache is its peculiar intolerance to alcohol, and to some extent tobacco. I have noted, when a patient is fairly comfortable and decides that a hot drink before retiring will benefit his condition, he is in-

variably made much more uncomfortable. This is, of course, due to the congestion which the alcohol causes in the head adding to the already swollen and inflamed mucosa, and its becoming suffused to the point of engorgement.

One more point of importance is the subjective localization of the pain. In a given sinus infection, the pain will always recur in the same place, and while that sinus alone is involved, it will remain constant. This holds good even during the acute exacerbations of the inflammatory process, the pain, of course, reacting accordingly. If, however, the infection spreads to a neighboring cavity, the pain may then assume the characteristics pertaining to the latter, as well as continuing in the old locality. This will be further elucidated when treating with the sphenoid.

Regarding the complete absence of pain in attacks of sinusitis that are worthy of the name, I am somewhat of a skeptic. Certain authors (notably continentals) have reported such cases, but I have never met one in my own practice in which either pain or tenderness had not been present. The experiences of my own colleagues seem to bear mine out in this matter. This, of course, refers to infections with suppuration, and does not include those mild hyperplastic conditions which often affect the ethmoid.

Let us now apportion the head into areas corresponding to those usually affected by the individual sinuses. This is shown by the accompanying illustration:

The confinement of the pain within these specified areas, as attributed to individual sinuses, is not invariably accurate, but bases itself quite well for purposes of differentiation. For example, headache from frontal sinusitis is usually confined to the supraorbital region, but may, in certain cases, extend well into the parietal area, a region usually dominated by the posterior ethmoid and sphenoid. Again, the typical sphenoid head pain is in the occiput, yet I have seen cases where it was transmitted to a spot directly over the front of the maxillary sinus on that side. By and large, however, we can accept the diagrammatic illustration as fairly illustrative of the typical head pains as caused by the different sinuses.

Let us now consider our subject from the standpoint of the individual sinuses, only chronic infections being considered, as time is lacking, and, roughly speaking, the acute infections,

with the possible exception of the sphenoid, are more or less self evident.

The Maxillary Sinus.

Paradoxic as it may seem, the typical pain from an inflamed antrum is directly over the frontal sinus on the same side. This may be so severe and persistent as to simulate a frontal infection, and, indeed, many a frontal sinus has been chiseled open and found to be healthy on this very account, a circumstance which some time ago occurred in our city. One would expect, in a case of sinusitis, that no matter where the pain radiated, at least it would be particularly marked over the limits of the sinus cavity and, even if quiescent, could be elicited by means of pressure or tapping. Only very exceptionally does this occur in chronic maxillary sinusitis. Occasionally, during an acute exacerbation, the patient will complain of a feeling of pain in the antrum, but on close questioning it will be found that the sensation is not so much an ache as a feeling of distention or pressure. Tenderness on even deep pressure is not elicited.

The Frontal Sinus.

Chronic inflammation of this cavity always seemed to me to be analogous to chronic appendicitis. The two run almost identical courses, periods of quiescence interspersed with acute exacerbations, no one ever knowing just when a flare up will occur and how serious it will be. Usually headache associated with this in the stage of quiescence depends considerably upon the size and extent of the sinus, but presuming the cavity of normal area and the infection not particularly virulent, the pain is of a dull character, not quite sufficient to keep the patient confined to the house, but enough to interfere seriously with his business pursuits, particularly brain work, such as an accountant or bookkeeper. He will say, "Doctor, this headache annoys me so I can't keep my mind on my work." In other words, he has lost the ability to keep the attention concentrated. The pain usually remits some time during the afternoon, often completely, but in bad cases seldom entirely. When an acute exacerbation occurs, the headache augments corresponding to the swelling and consequent pressure that obtains within the sinus cavity. This is when we have those

pains which are of a splitting or throbbing character, in which the reaction of the patient is one of intense suffering. Indeed, they may become so unbearable as to awaken suicidal tendencies in our weaker brethren. As the acuteness subsides, the pain also recedes, until it reaches its lowest ebb, a degree of more or less relative comfort, which in that particular case may be considered normal.

The entire frontal sinus may not necessarily be affected in order to have pain of this character. Indeed, I recall a patient who suffered so greatly, that she had long since been obliged to forego all social functions. Operation exposed a completely healthy frontal sinus, but a finger like, partially enclosed cell on the posterior or cerebral wall, about the diameter of a small lead pencil such as seen on dance programs, was found to be filled with purulent granulation tissue. This was eradicated, and the pain has not recurred from that day to this.

Curious as it may seem, the opposite may also obtain, and the pain may not necessarily be severe proportionately to the duration of the disease. In other words, it does not necessarily follow: The more chronic the disease, the greater the headache.

I am reminded particularly of a young man who came into the hospital after a peculiarly severe attack of acute frontal sinusitis from deep diving. He presented himself for diagnostic purposes, feeling much improved over his condition of the previous week, and rather resented the idea of an operative procedure; but it was insisted upon, and at the time of operation, at least half of the frontal lobe was covered by an extradural abscess.

It is interesting to note that even beginning cerebral complications need not necessarily be accompanied with severe local manifestations, particularly when the silent area of the brain is involved.

The Ethmoid.

This structure is unlike the true sinuses, as they represent cavities situated outside of the nasal fossa, while the ethmoid labyrinth is a mass of cells of a spongy consistency lying within the nasal chambers. Under these circumstances, we can rightly suppose that head pains from disease of this structure

would be of a different character than those from the true sinuses, surrounded by hard, unyielding bony walls. The ethmoid labyrinth is subject to two distinct forms of infection: Chronic hyperplastic, with polyp formation, and chronic suppurative. In both of these forms of the disease, headache is one of the most inconstant symptoms, the violence of the pain having apparently no relation to the severity of the disease. In certain cases, the pain will be quite severe, yet the actual pathologic changes are insignificant; in others, the headache is mild, yet enormous tissue changes have taken place within the sinus. While a special treatise on the various ethmoid headaches would require the two forms to be dissociated, in this instance, lest we become too involved, it might be wise to consider them both under one heading. As noted in the diagram, headache from ethmoid inflammation is felt particularly over the parietal regions. A dull pressure would perhaps describe it better than an actual pain. This, of course, varies at even different hours thru the day and, when severe, sometimes extends into the temporal regions. In severe cases, even after the pain leaves, the scalp remains sensitive to pressure. As the infection is apt to be more advanced on one side of the ethmoid capsule, the pain will naturally be more pronounced on that side. Regarding a differentiation between the pain in the polypoid and suppurative varieties, there is little to choose. Broadly speaking, however, one can say that in the hyperplastic type it is more constant, while in the suppurative, it is apt to be much more severe.

Another point of distress in ethmoid infections is between the eyes, directly above the root of the nose. The patient says there is a feeling as tho a wedge were prying things apart, and if he could only get his fingers in there he could pull it out. In other words, patients can localize the seat of their pain within very small limits. This is by no means constant, but when present is pathognomonic.

The Sphenoid Sinus.

Pain from this, the deepest lying sinus cavity in the cranium, is perhaps one of the most overlooked, and yet most signficant of all those head pains with which we are concerned. The headache usually associated with sphenoiditis of the chronic type is perhaps more typical than any of the others, possibly excepting the frontal. It consists of a diffuse feeling

of pressure in the occiput, sometimes extending into the mastoid processes and even into the temporal region. Extension downward into the shoulder of the affected side, the socalled sphenopalatine syndrome, popularized by Sluder, is also commonly associated with this affection.

The degree and extent of the pain is concomitant with the amount of pressure within the sinus. As this pressure, except in extreme instance, is not constant, it naturally follows that the headache must occur in periodical attacks, the severity of which is dependent upon the degree and prolongation of the secretory pressure. This may be caused, on the one hand, by the contained and continually forming secretion, and on the other by the approximation of the sides of the inflamed mucosa. Both of these forms may be exerted coincidently. As the pressure is relieved, the pain remits in intensity, the remission being directly as the length of time required for the passing of the congestion. During the paroxysms, or stage of retention, the cephalalgia changes its character to an intense sickening and throbbing, synchronous with the heart beat, while during the stage of quiescence, it assumes more the character of a heavy pressure upon the vertex. The periodicity of recurrence is not typical as in the frontal, but seems to come on at any time of the day, and does not remit with that abruptness as is so often noted with the latter.

By and large, however, a persistent occipital headache, especially if confined to one side (laterally), when recurring day after day with the utmost regularity is, if not pathognomonic, highly suspicious of a sphenoid involvement, and steps should immediately be taken to prove or disprove this assumption.

Another curious phase of the pain is its occasional appearance directly below the eye, in the region of the infraorbital nerve.

Dizziness, especially on stooping, is more frequently associated with pain from the sphenoid than from any of the others, and when marked is rather indicative of inflammatory disturbances within this cavity.

And now, gentlemen, I am done. In conclusion, however, it would seem that headache is one of the most common of all symptoms associated with a chronic sinusitis, and that the time, place and degree of these head pains, properly inter-

preted, despite their vagaries, constitute one of the most reliable signs in the symptomatic diagnosis pertaining to sinuology.

DISCUSSION.

Dr. Robert C. Lynch, New Orleans: I agree with practically everything Dr. Skillern has said, and I can best use my time by speaking of some situation which he did not touch upon or lay much stress upon. The first one is, that in vacuum headache, there is a difference between what might be termed an acute vacuum and a chronic vacuum. Acute vacuum is the situation in which the pain occurs as the Doctor has described—when the nasal blocking gives rise to those symptoms which we recognize in acute rhinitis in which the sinus has not become involved. In the chronic cases—I have seen about twenty, particularly in the antrum —the natural opening has been closed, and so the pain is not periodic at all, but is increasingly continuous. By that I mean, that as the days go on, the pain seems to be aggravated and is relieved only by opening the sinuses. The minute the sinus is open, the pain disappears and stays in that state until the sinus closes again, when the pain begins to appear, increasing in character as times go on, until the sinusitis develops. I should also like to mention one point Dr. Skillern made in regard to chronic sinus condition in which the pain is more of a toxic character, due to absorption rather than to pressure within the sinus. In these cases, with the sinus well open, drainage and ventilation having been established to an amount which would seem more than reasonable, sufficient to bring the mucous membrane back to its normal state, there will still be a continuation of low grade pain. It is not severe, but continuous, apparently a toxin, and is only relieved when the whole cause of the pathology is removed. I have seen some cases of sinusitis, with a definite pathologic change in the mucous lining of the sinus, without any pain whatever. However, the patient may have had pain early in the disease, and I think that is what Dr. Skillern referred to. The sinus is in a state that we are sure the patient must have had some pain, but frequently they come under observation when there is no symptom of pain, but the mucous membrane has undergone a pathologic change.

I also would like to mention the closed sinus, mucocele, in which as long as the walls are not broken down, we have an infection, and as soon as the wall is broken thru we have a cessation. In other words, it represents the same situation as a decompression operation would do.

I would also like to call your attention to the fact, that in my experience, I have had five cases in which there has been crossed pain. I would like to know whether any other member has had this situation. All five patients complained of left supraorbital pain, with the right maxillary sinus involved. Upon cocainization of the inferior meatus for puncture into the antrum, when puncture of the right antrum was made, the patient complained of left supraorbital pain. Washing the right antrum caused pain in the left supraorbital region.

Dr. William C. Bane, Denver: In considering head pains as symptomatic of accessory sinus disease, we must bear in mind that there is a

small percentage of individuals who do not experience ache or pain from abnormal conditions, while, on the other hand, a large percentage of patients with accessory sinus disease, especially in the acute stage, as with errors of refraction, have more or less headache due to nerve irritation. The headache in some sinus cases is due to the action of the toxin. In a large proportion of the cases of acute frontal sinusitis, there is manifest the socalled "sun pain" that appears during the forenoon and ceases in most patients during the afternoon, to return about the same hour the following forenoon. In the most severe cases, the pain comes on early and continues for a greater number of hours. In this class of cases, tenderness is, as a rule, found upon pressure above the inner canthus and on percussion over the brow. Occasionally we see a patient with pain in the eye and inability to use the eyes with comfort, the real cause of complaint being disease of the adjacent frontal sinus. Two days ago, I was asked to see a patient that one week previously had sustained an injury to his right eye, a superficial cut on the sclera by a bit of rock. He was cared for by Dr. Black, the wound healing promptly with vision of 20/16. Examination did not reveal any congestion of the eye, but marked tenderness of the frontal sinus over the eye. He had had pain over the eye daily for three days, coming on about eleven in the morning and continuing until evening.

Rarely have I seen a case that I recognized as the socalled vacuum frontal headache described by Sluder, in which the socalled Ewing's sign is present, that is, tenderness above the inner canthus and discomfort upon using the eyes. In acute inflammation of a maxillary sinus, very rarely is tenderness elicited upon pressure, but there may be occipital pain complained of. The pain of ethmoidal involvement is deep between the eyes, a pressure pain. As to the infection of the sinuses, the influenza bacilli are the most constant in accessory sinus suppuration. The diplococcus, pneumococcus, staphlyococcus pyogenes, albus and aureus, and streptococcus pyogenes are also found in the sinuses. Patients with lowered resistance, and those with tuberculosis, are the most difficult cases to cure.

Dr. Ross H. Skillern, Philadelphia (closing): As to the periodicity of these headaches I think that can be explained in this way. When the patient reclines during the night, the blood pressure is equalized thru the entire body, and the sinus mucosa becomes more engorged than when the patient is in an upright position, so when reclining there is complete engorgement of the sinus mucosa. As he starts to walk around, the secretion begins to gravitate and finds its way thru the engorged tissues down into the nasofrontal duct and plugs it up, thus causing a headache. After a time perhaps in two to five hours, the air begins to work its way thru the nasofrontal duct, and finally gets into the sinus; then the duct becomes opened and the headache instantly disappears. How often patients come to you and say, "Doctor, this headache will go on all morning and then suddenly disappear, and about a half-hour afterwards I blow the worst looking stuff out of my nose." Of course, that is because the purulent material works down until it collects in a large enough mass to be blown out. It seems to me, that is the best solution of these periodic headaches.

POLYPOID DISEASE OF THE ANTRUM.

EDWARD KING, M.D.

CINCINNATI, OHIO.

It seems best to me to confine my remarks in this short paper to a few observations on the maxillary antrum. There is a rather well founded belief among the laity, that one nose operation begets a second, and from there on ad infinitum. No doubt this is owing to the tendency of nasal polypi to recur. Very often, in recommending a nasal operation to one of our patients, we hear the story of a relative who has had innumerable operations on the nose, without very much relief. We endeavor to explain the nature of the disease, and to excuse our lack of measures for eradicating it, but, unfortunately, we are in no position to assure the patients who come to us for relief from nasal polyposis that the disease will not reappear. In spite of the most painstaking work, we do find recurrences, hence it is necessary to give each case the most thoro study, in order to diminish the number of failures so far as lies in our power.

Repeated attacks of inflammation in the nose and the extension into the sinuses give rise to chronic inflammatory changes in the sinus mucosa, resulting in thickening of the mucous membrane and pressure on the deeper layers containing the glands. The mucous membrane becomes edematous, and the glands become cystic. The antrum is particularly susceptible to these changes, because of its position. No doubt a large middle turbinate which hugs the lateral wall is an important factor in the causation, because it prevents aeration and drainage. In acute suppurating sinus infections, the antrum is the last to drain. On numerous occasions, it has been my experience to find large quantities of pus in the antra following an acute cold, after all symptoms had disappeared and the nose showed no signs of pus. To my mind, it is not difficult to conceive of chronic changes in the antrum without very marked signs in the nose.

As a rule, the hyperplastic changes which give rise to

symptoms occur in adults. However, we have had some experience with polypoid changes in children, and no doubt, as our knowledge of sinus disease in childhood increases, we will have more occasion to see these conditions.

The details of three cases which I have had the opportunity to study may be of interest in this connection. The first is that of a child, age 8, who gave a history of chronic suppurating sinus disease following an attack of measles at age 3. The operation revealed polypoid changes in the ethmoids and antra. The ethmoids were removed, and a very large opening made under the inferior turbinate in both antra. The most careful aftertreatment was given, with a great improvement in the child's health, but I am afraid that the antra will not respond without more radical surgery.

The second case is that of a boy, age 10, who gave a history of vasomotor rhinitis since age 5. The tonsils and adenoids and a small piece of the middle turbinate on both sides had been removed at a previous operation. Examination revealed polypoid degeneration of the middle turbinate and the ethmoid cells. X-ray of the sinuses revealed cloudy ethmoids and antra. The middle turbinates and ethmoids were removed, and an opening made into the antra thru the inferior meatus. Complete relief from all symptoms was the result for about 18 months, when the condition recurred gradually. Examination revealed polypi in the nose, and the openings into the antra completely closed. The X-rays again showed cloudy antra, and opening thru the canine fossa was recommended. The family refused to submit the child to any more surgery, at least at my hands.

The third case is that of a boy, age 14. His only symptom was difficulty in breathing. The history revealed that he had been operated for nasal polyps on two previous occasions. At age 9, a large mass of polyps had been removed from the left side of his nose. He was operated again at 12 years of age for the same thing. Examination of this boy showed a large polyp completely occluding the anterior nares. Postrhinoscopy revealed a number of polyps retronasally. The X-ray showed marked clouding of the left antrum. At operation, the antrum was opened in the canine fossa, and a large mass of polypoid material was found filling the cavity. Some of this mass extended into the nose. This boy has been free from polyps for a period of over two years. An analysis of these cases

points to the fact that marked hyperplastic changes are found early in childhood, and that the antrum is frequently involved.

The diagnosis of well marked cases of hyperplasia does not offer many difficulties, but in a study of these cases and in recommending an outline of treatment, it is necessary to consider the antrum. The removal of polyps is rather simple, and the relief is so prompt, that we may approach the operation without careful consideration of all the factors. After thoroly removing every vestige of polyp, exenterating all cells

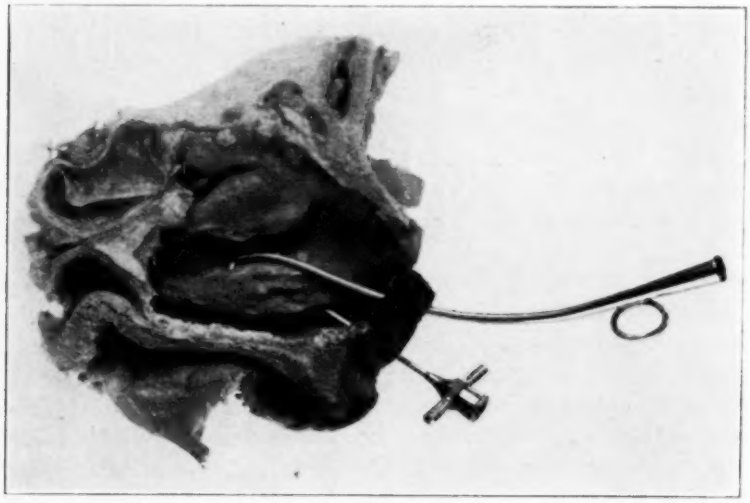

Figure 1. The relative positions of the ordinary straight antrum needle and the cannula described in the text. The cannula has entered an accessory ostium.

and opening the sphenoid, it is rather discouraging to have the patient return in a short time with the nose full of polyps again. I believe, that when the X-ray shows clouding of the antra in the presence of nasal polyps, the antra should be opened thru the canine fossa.

The less marked and mild degrees of hyperplasia demand constant and continued study. There may be very slight changes noted on inspection of the nasal cavity. The middle turbinate may be normal. The ethmoid must be inspected carefully. To make the examination, it is necessary to infract the middle turbinate and examine the ethmoid floor and lateral nasal wall, while holding the turbinate over toward the septum. The nasopharyngoscope may show mild hyperplastic

changes around the antrum opening. In vasomotor rhinitis, it has been my experience that the signs may be present at one examination and absent at another, so that repeated examinations may be necessary. A grayish exudate under the middle turbinate, which gives me the impression of a damp ethmoid, may be seen at times.

The diagnostic irrigation should be made in all cases. This procedure should be carried out thru the middle meatus. Too much stress cannot be laid on this method of washing the

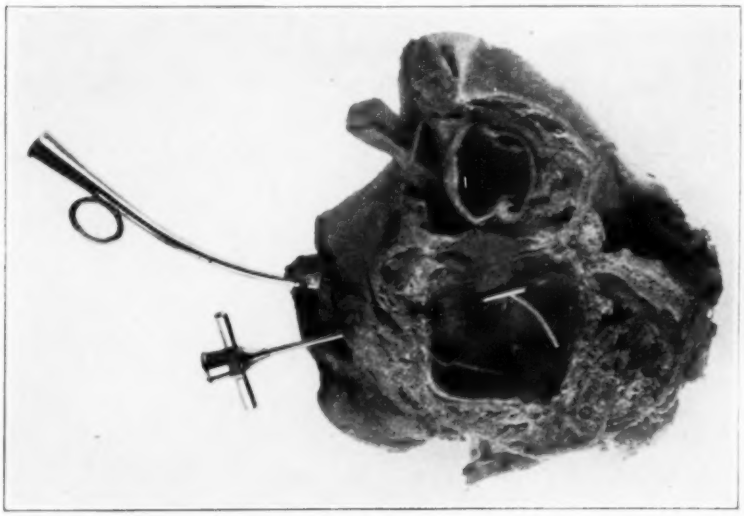

Figure 2. A view of the instruments in the antrum, the external wall of antrum having been removed.

antrum, because of the ease with which it is performed. The cannula, which is to be presented for your approval, is designed to enter the middle meatus under the middle turbinate, so that there is never any necessity for disturbing this structure. It is firm enough to pass thru the thin membranous partition with ease, if the natural or the accessory opening cannot be located. The bore of the cannula is large enough to permit a stream of water as great as the ordinary diagnostic needle. In practice, the upper surface of the inferior turbinate and the lateral wall of the middle meatus are cocainized with 20% cocain on a fine bent cotton carrier. The cannula is then employed as a probe to feel for the opening, and is inserted in the most favorable spot. If there is any doubt as to its loca-

tion, a bent probe may be used to verify the position of the opening. This method has none of the disadvantages of antrum puncture and should be used more generally.

X-rays of the sinuses must be made in all cases. The interpretation of the pictures rests on the clinical findings. With the presence of polyps in the nose and cloudy antra on X-ray, the antra should be opened. Lipiodol has been of value in these cases, because it outlines the polypoid mass. Two slides

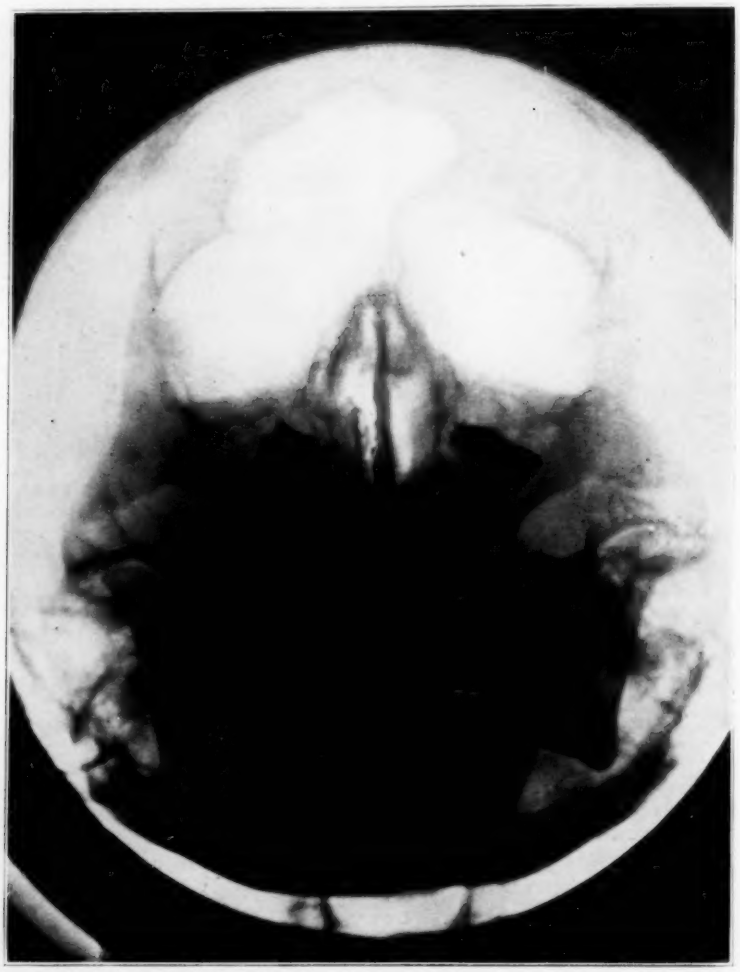

Figure 3. The above illustration shows the dipping in effect of the lipiodol in a case of marked hyperplastic maxillary sinusitis.

showing the lipiodol surrounding and outlining the tumor mass are to be shown.

Where the X-ray shows slight clouding, or the picture is negative, we must depend on the clinical findings and experience for the course to pursue.

In doubtful cases, it is advisable to make a window in the facial wall of the antrum, in order to make a direct inspection of the mucosa. A nasopharyngoscope or a small mirror can

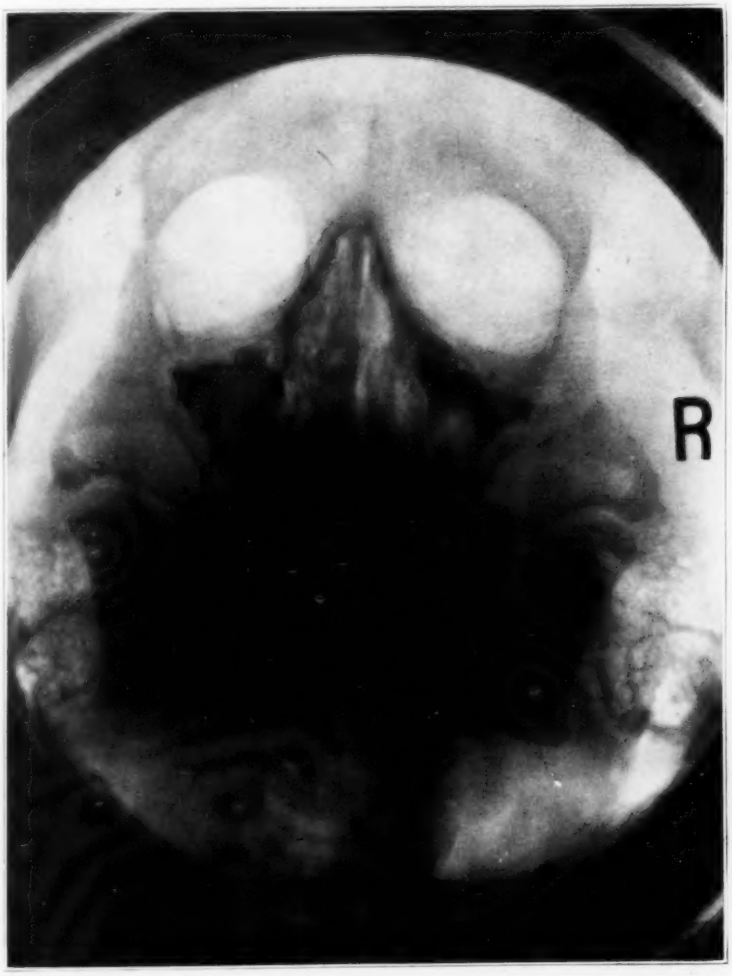

Figure 4. The above illustration shows the lipiodol filling the antra and surrounding the tumor mass, which is indicated by the lighter areas.

be inserted thru this opening for a survey of the entire cavity. Experience with this procedure has taught me, that very often when we would be inclined to give the antrum a clean bill, or to make an intranasal opening and hope for a good result, the small window has disclosed pathologic changes which warrant only a radical antrum. It is simple to make the opening, and if the antrum is clear, then the mucous membrane can be closed without having done any damage.

DISCUSSION.

DR. JOHN J. SHEA, Memphis, Tenn.: Sinus disease is progressive in nature, and the operative cure of one sinus or of a group of sinuses is no insurance to the remaining sinuses that they are immune. Operative procedures as applied to the nasal sinuses are for the removal of some disease present, and only those measures that correct faulty ventilation or drainage have any influence on the future. Heredity is a factor to be considered, and an adult sinus reflects in its development the infections that it has suffered during its formative period. Diet and susceptibility to suppurative disease influence the frequency of acute suppurative sinusitis. Therefore, a patient who suffers from sinus disease sufficiently to require an operation will as a rule require subsequent operations on other sinuses, and many of them have in turn all the sinuses operated upon by one or more surgeons. Finally, when all the sinuses have large openings into them but still are diseased, I suggest the use of radium for the treatment of the reformed tissue.

I agree with Dr. King in several of his statements, and wish to endorse the idea of irrigating the antrum thru its natural opening. There are two drawbacks to this procedure, you miss the dental cysts, and secondly, the patient often struggles and you lose your specimen for study. I wash thru this area in all treatment cases, but for explorative punctures use the same technic in the adults as Dean uses in the children, and allow the washings obtained in the syringe to stand in test tubes. By cooling the washings, physical changes often occur, and what appeared at first to be a normal washing turns out to be pathologic. Likewise, slight changes of the chemical reaction will bring out the pathologic nature of the washings. Like Dr. King, I am an advocate of roentgenograms and offer this idea to those who have not been obtaining help from their plates: Take several plates of lighter penetration than the usual dark picture, for the dark plate reveals only details of the skull and does not record the changes in the sinus membrane, and with this technic, pathology will be demonstrated in the sinuses. I further substantiate Dr. King, in that chronic pathology in the antrum should be inspected, and if found chronic, should be removed radically. It is marvelous the amount of pathology found in some antra which have been constantly irrigated or drained intranasally. Likewise, chronic ethmoids and frontals which have resisted intranasal surgery and are the foci of infection should be dealt with radically. The more adept we become at radical procedure in adults, the more permanent are our results, but I do not sanction radical sinus surgery in children.

PERORAL ENDOSCOPY IN ITS RELATION TO OTOLARYNGOLOGY WITH SPECIAL REFERENCE TO POSTOPERATIVE COMPLICATIONS.*

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Peroral endoscopy, including direct laryngoscopy, bronchoscopy, esophagoscopy, and gastroscopy, constitutes a special field in medicine, and requires for their safe and efficient performance special training and the skill that comes from long continued practice in working thru a tube with one eye only. The otolaryngologist is accustomed to monocular vision, and very quickly acquires the ability to correctly interpret the picture seen thru the endoscopic tube. He is also familiar with the landmarks of the pharynx and larynx, and is accustomed to operative work and the special technic that is required in the upper air and food passages. The first requisite in the work is an adequate instrumentarium. Small tubes are required for infants and longer tubes for adults. In short, the tubes must be adapted to the size of the passages to be examined, and the accessory instrumentarium must be proportionate to the size of the tube. There is no universal endoscopic tube. The adult instrument will kill the infant without accomplishing the desired examination. The length of the instrument used in the infant would not accomplish the desired result in an adult; similar small diameters are required to enter peripheral bronchi in the adult, but the length must be adequate to reach the periphery of the adult lung. Notwithstanding these well known facts, there have been reported in the past a number of cases in which attempted examination of the child with an adult tube has been followed with fatal results.

Endoscopy is most safely and most successfully performed without anesthesia, or with local anesthesia only, and to carry it out with safety and precision an edoscopic team, thoroly

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drilled in the technic of the work, is necessary. This will best be accomplished by two or more endoscopists working together.

However, it would seem necessary that every laryngologist should be prepared to examine the larynx of patients of any age; in very young children, the mirror is useless for this purpose. The diagnosis of laryngeal diphtheria may depend on the taking of a swab, for smear and culture, directly from the larynx, and direct laryngoscopy is the only means by which the larynx of infants and very young children may be examined. Again, differential diagnosis of an ulcerative lesion of the larynx may necessitate biopsy. This can be most accurately done by direct laryngoscopy.

The diagnosis of laryngeal stenosis may call urgently for bronchoscopy. Whether or not the otolaryngologist shall prepare himself for bronchoscopy for foreign body and disease, is a question that each individual must decide for himself. A laryngeal foreign body may be easily converted into a bronchial or esophageal foreign body by attempts at removal and so require bronchoscopy or esophagoscopy. The internists and the thoracic surgeons have come to regard blind methods in the diagnosis of esophageal disease as obsolete, because at best such methods can be only inferential, and are always extremely dangerous; for consultation and direct inspection of the esophagus, they naturally turn to the otolaryngologist for esophagoscopy.

An infective, postoperative pulmonary condition may call urgently for bronchoscopy; or a foreign body accident during an otolaryngologic operation may demand a bronchoscopy or esophagoscopy. The otolaryngologist who is trained and expert in peroral endoscopy will be able to care for the postoperative complications that may occur in his own practice, tho, under these circumstances, he may prefer to have responsibility taken by a confrere. In this connection, I should like to present a group of cases in which foreign body accidents occurred during otolaryngologic operations. In over seventeen hundred and fifty foreign body cases that came to our Clinic, there have been fourteen cases due to accidents during otolaryngologic procedures. When we consider the enormous number of operations that are done upon the nose and throat every year, it is a great tribute to the careful tech-

nic and skill of otolaryngologists that these accidents do not occur more frequently.

Case 1. Foreign Body No. 649. *Lamp from Whitehead Mouth Gag in Right Bronchus.* A boy, ten years of age, during tonsillectomy under ether anesthesia, in the dorsal recumbent position, inspirated the small lamp from a self illuminated Whitehead mouth gag. The day following operation, fever and cough developed, with evidence of obstruction of the right bronchus. Three days after the accident, the temperature was 103°, and physical examination showed complete obstruction of the right lung. Roentgen ray examination showed the lamp in the right main bronchus, with complete atelectasis of the right lung, the heart and mediastinum being displaced to the right. Forty-eight hours after the bronchoscopic removal thru the mouth, the atelectasis had disappeared, the temperature was normal and recovery was complete.

Case 2. Foreign Body No. 901. *Catch from a Tonsil Forcep in Right Bronchus.* A boy, seven years of age, during tonsillectomy under ether anesthesia, in the dorsal recumbent position, inspirated a catch that broke off the tonsil grasping forcep. Physical examination gave evidence of foreign body in the right lung. Roentgen ray examination showed a portion of tonsil forcep in the right lower lobe. Peroral bronchoscopic removal eighteen days after the accident was followed by perfect recovery. The foreign body being metallic and not completely obstructing the portion of lung in which it lodged, did not give rise to serious pathologic change during its sojourn.

Case 3. Foreign Body No. 1582. *Tonsil Snare Wire in Right Bronchus.* During tonsillectomy under ether anesthesia, in the dorsal recumbent position on this boy, twelve years of age, the tonsil snare wire broke and a portion of it disappeared in the pharynx. Inspection of the chest the same day showed limitation of expansion over the right lower lobe. Other physical signs of foreign body were negative. Roentgen ray examination showed the piece of snare wire, about 2½ cm. long, in the right lower lobe bronchus. On peroral bronchoscopic removal, there was found to be no evidence of abnormality in the bronchus. The wounds in the tonsil fossae healed normally.

Case 4. Foreign body No. 1545. *Gold Tooth Crown in Right Bronchus.* A woman, 51 years of age, had a tonsillectomy performed under ether anesthesia, in the dorsal recumbent posi-

tion. A few days later, she developed a cough with wheezing respiration. Pulmonary symptoms persisted, and it was thought that she had developed bronchial asthma. For two and a half months, her condition did not respond to treatment. The patient had missed the gold crown from a molar tooth following the operation, and suggested it might be in her lung and the cause of her asthma. In clearing up the possibility of aspiration, Roentgen ray examination showed the gold crown in the right bronchus. The patient on admission had no complaints except slight cough and a marked asthmatoid wheeze. After peroral bronchoscopic removal, the wheeze disappeared immediately and the cough and asthmatic symptoms entirely disappeared.

Case 5. Foreign Body No. 1466. *Deciduous Tooth in Right Bronchus.* A girl, eight years of age, who was well prior to tonsillectomy under ether anesthesia, in the dorsal recumbent position, developed cough and fever with signs in the right chest, on the second day following operation. Roentgen ray examination showed the crown of a deciduous tooth in the right lower lobe bronchus. On her admission, ten days after aspiration of the tooth, she was running a slight fever, and there was evidence of retention of secretion in the right lower lobe. Peroral bronchoscopic removal of the tooth was followed by complete recovery. Patient was discharged from the hospital on the third day after bronchoscopy.

Case 6. Foreign Body No. 1530-C. *Deciduous Tooth in Right Bronchus.* A boy, ten years of age, had a tonsillectomy done under ether anesthesia, in the dorsal recumbent position. He developed cough, pain in the right chest, and fever immediately after the operation. He went home under the care of the family physician, and was supposed to have developed pneumonia. When the symptoms did not clear up at the end of three weeks, the otolaryngologist insisted on Roentgen ray examination of the chest. There was found the crown of a small deciduous tooth in the right bronchus, with evidence of retained exudate in the middle of the lower lobes. The child, on admission to the Clinic, was running an irregular fever and was quite toxic. Peroral bronchoscopic removal of the tooth by Dr. L. H. Clerf was followed by complete recovery, the child leaving the hospital on the third day following the removal of the tooth.

Case 7. Foreign body No. 1654-C. *Deciduous Tooth in Left Bronchus.* A boy, eight years of age, following tonsillectomy

under general anesthesia, in the dorsal recumbent position, developed irregular fever with cough and expectoration, two days after operation. There had been five teeth extracted at the time of the tonsillectomy, but these were accounted for by the operator. Roentgen ray examination, eight days after the tonsillectomy, showed a deciduous tooth in the left bronchus. The tooth had been loosened by the mouth gag. Dr. McCrae reported on physical examination, that there was evidence of blocking of the left bronchus, with some atelectasis and retention of secretion in the left lung, marked limitation of expansion on the left side. Peroral bronchoscopic removal, by Dr. L. H. Clerf, was followed by prompt recovery, the child being discharged from the hospital the fourth day after bronchoscopy.

Case 8. Foreign Body No. 1591. *Deciduous Tooth in Right Bronchus.* Boy, eleven years of age, on whom tonsillectomy was performed under ether anesthesia, in the dorsal recumbent position, developed an irritative cough and wheezing respiration 24 hours after the operation. He was discharged from the hospital and came under the care of the family physician, who thought he had caught cold, and that his symptoms were due to enlarged mediastinal glands. His cough was unproductive. He was kept under observation for two weeks. The otolaryngologist finally succeeded in having a Roentgen examination of the chest, which revealed an opaque foreign body in the right main bronchus. The internist felt that the ray examination was not necessary. On his admission to the Clinic, the child did not appear ill, and the wheeze and nonproductive cough persisted. Physical examination by Dr. Perry Pepper localized a foreign body in the right main bronchus, with evidence of emphysema of the right lung, with displacement of the heart to the left. Roentgen ray examination by Dr. Pancoast showed a foreign body, the shape of the crown of a deciduous molar, in the right main bronchus. On bronchoscopic examination, it was found that the tooth was in such a position at the orifice of the right upper lobe bronchus, and was so surrounded by granulations, as to allow admission of air to the right lung on inspiration and prevent its escape on expiration, thus accounting for the most unusual picture for this type of foreign body, of obstructive emphysema in the portion of lung distal to the foreign body. After peroral bronchoscopic removal, recovery was complete, the patient being discharged well two days after bronchoscopy.

CASE 9. Foreign Body No. 1729. *Deciduous Tooth in Right Bronchus.* A girl, nine years of age, was admitted to the Clinic one month following tonsillectomy under ether anesthesia. Following the operation, she developed a dry, unproductive cough, and also noticed that a tooth had been lost. The missing tooth was not found. No Roentgen ray examination was made at that time. Child was sent home and cough continued. She developed fever and loss of weight. Three weeks after tonsillectomy, the cough became very productive, the sputum consisting of foul, blood stained pus. Roentgen ray examination was then made, which revealed a deciduous tooth in the right stem bronchus. The tooth was removed bronchoscopically without anesthesia. Patient made a complete recovery.

CASE 10. Foreign Body No. 1540. *Tonsil Sponge in the Left Bronchus.* A man, forty-four years of age, was operated upon under local anesthesia, in the upright position. A turbinotomy and submucous resection of the septum had been done, and the nose packed. The left tonsil had been removed, and a gauze sponge saturated with 10% cocain was packed in the tonsil fossa. The right tonsil was being injected, when the patient gagged and choked, and inspirated the sponge from the fossa. The right tonsil was removed and the chest examined, and it was found that air did not enter or leave the left lung. The bronchoscopist was called in consultation, and on physical examination we found the left bronchus completely blocked. Roentgen ray examination confirmed this finding. On peroral bronchoscopic examination, the left bronchus was found blocked by blood clot and the tonsil sponge. The blood clot and tonsil sponge were removed, and the tracheobronchial tree found normal. The man made an uneventful recovery from the nasal and tonsil operation.

CASE 11. Foreign Body No. 1464. *Nasal Rasp in Left Bronchus.* A man, forty-five years of age, during a frontal sinus operation under general anesthesia, in the dorsal recumbent position, inspirated a portion of a rasp, which had broken while being used to enlarge the opening into the frontal sinus. In order to locate the missing portion of the instrument, a Roentgen ray examination was made the following day, which showed a portion of a Brawley's rasp, ¾ of an inch long, in the left bronchus. The only sign of foreign body was Dr. McCrae's sign of limitation of expansion on the invaded side. Recovery was uneventful after bronchoscopic removal of the for-

eign body. The expansion of the left side of the chest became normal.

Case 12. Foreign Body No. 679. *Intubation Tube in Left Bronchus.* A boy, seven years of age, was admitted to the Hospital with a diagnosis of lung abscess. Roentgen ray examination showed an area of pathology in the left lung surrounding a shadow of an intubation tube. The history elicited was, that eighteen months previously, the child had had laryngeal diphtheria, and during intubation a tube had been lost. It was supposed to have been swallowed and passed in an unwatched stool. The child had developed pulmonary symptoms within a month of the recovery from diphtheria. These had continued up to the time of his admission to the hospital. The tube was of the O'Dwyer type, and the number one size, and had been used in a six year old child. This accounted for the fact that the tube passed thru the glottis. If the proper size tube had been used, the accident could not have happened. The foreign body was removed bronchoscopically without anesthesia, and the patient recovered completely.

Case 13. Foreign Body No. 1518-T. *Wooden Applicator and Swab in Right Bronchus.* A girl, 22 months of age, developed an attack of acute follicular tonsillitis. After several days illness, the father, who is a physician, attempted to take a culture from the throat with a wooden applicator. The stick broke and the child inspired the cotton wrapped end of the stick. The child had no anesthesia and was in the upright position. Roentgen ray examination made by the father the next day showed atelectasis of the right lung. The child was brought to the clinic five days after the inspiration of the swab. She was markedly dyspneic on admission, and Dr. McCrae on examination believed the foreign body to be in the right bronchus and trachea. Roentgen ray examination, by Dr. Manges, showed an atelectasis of the right lung, with evidence also of tracheal foreign body. On peroral bronchoscopic examination, a large amount of very foul pus was found and aspirated from the trachea; the swab was found in the right main bronchus with the stick projecting into the trachea. After removal of the swab, the convalescence was prolonged. It was necessary to aspirate the secretions bronchoscopically at frequent intervals. The child was discharged well at the end of three weeks. The violent inflammatory reaction in this case was due, we believe, to, first, the character of the

foreign body, the cotton being a vegetal substance; second, the culture from the tracheal secretion showed a virulent growth of Friedlander bacillus.

Case 14. Foreign Body No. 1712. *Radium Capsule in the Stomach.* A boy, thirteen years of age, had an operation for sarcoma of the antrum, followed by radium application. Two radium containers covered with a finger cot were placed in the antrum, fastened by a string. At the time for the removal of the radium, it was found missing. Roentgen ray examination showed the radium to be in the boy's stomach. The radium capsules were removed by gastroscopy with double plane fluoroscopic aid. There were no untoward effects from the sojourn of the radium in the stomach or following the gastroscopy.

There is also another group of cases in which there is a postoperative pulmonary infective condition, that I should like to mention briefly. A very large percentage of the pulmonary suppuration cases that we are called upon to treat follow operations on the nose, throat, and mouth. Time will permit of a brief presentation of only 4 cases.

Case 1. *Posttonsillectomic Pulmonary Abscess.* The first case is that of a boy, seven years of age, who was referred to the Clinic ten days after tonsillectomy. The pulmonary symptoms had come on five days after the operation. On admission, there was involvement of his left upper lobe. Roentgen ray examination showed a large cavity, with a fluid level in the left upper lobe. He was treated bronchoscopically at weekly intervals, and after seven treatments was discharged well. Roentgen ray examination showed the lungs to be clear.

Case 2. *Posttonsillectomic Pulmonary Abscess.* A man, twenty-six years of age, developed a posttonsillectomic pulmonary abscess one week after operation. He was admitted to the Clinic three weeks after the onset of the pulmonary symptoms. The abscess was found to be in the left lung, posteriorly, at the level of the lower border of the scapula. Bronchoscopically, it was determined that the abscess was in the lower portion of the upper lobe, and not in the upper portion of the lower lobe. The location of the abscess was unfavorable for treatment bronchoscopically. He was referred to Dr. George P. Muller, who did a thoracotomy and drained the abscess. The man made a complete recovery.

CASE 3. *Posttonsillectomic Pulmonary Abscess.* A woman, thirty years of age, was admitted to our service at the University Hospital, one month following a tonsillectomy, with an infective process involving the middle and lower lobes of her right lung. Under bronchoscopic treatment, at weekly intervals, the infective condition cleared up, and the patient was discharged well after ten treatments. After six months, she gained thirty pounds in weight and is entirely well.

CASE 4. *Posttonsillectomic Pulmonary Abscess.* A man, twenty-five years of age, five days following tonsillectomy, developed a pain in his left chest, and blood stained purulent expectoration followed. One week after his operation, he was admitted to the bronchoscopic clinic for treatment. Roentgen ray and physical examinations showed involvement of the lower lobe of his left lung. On bronchoscopic examination, the posterior division of the left lower lobe bronchus was found inflamed and obstructed by blood stained pus. The secretion was aspirated and local medication applied. Treatment was repeated at four day intervals, and after eight treatments, the lower lobe bronchus was normal. The man was symptomatically well, had gained ten pounds in weight, and the bronschoscopic instillation of lipiodol into the area of lung that had shown an abscessed cavity gave us a graphic proof that the cavity had disappeared.

In the first two cases of this group, we believe the infection occurred thru the embolic route, and in the last two, the infection was probably inspiratory in origin. These cases will serve to illustrate the value of endoscopic procedure in this type of postoperative complication.

The enormous demand for careful and safe bronchoscopy for the diagnosis and treatment of pulmonary diseases is making serious inroads on the time of the otolaryngologist. There seems to be an increasing need in the large hospitals of the great medical centers for a fully equipped bronchoscopic clinic prepared to render safe assistance in the diagnosis and treatment of diseases of the lungs, esophagus, and stomach. The logical man for this work is the otolaryngologist, if he has the time.

DISCUSSION.

Dr. Harry L. Baum, Denver: The development of this specialty, of which Dr. Tucker is so distinguished an exponent, is comparatively recent, but has been in the last few years really more widespread than I think most of us would have thought possible some years ago. As a result, there has been a great demand for men trained in the particular manipulations required in doing this work. In most instances, it has fallen to the otolaryngologist to develop his technic and to do the bronchoscopies or the esophagoscopies that are necessary in his own community. Personally, I think there is probably no other man so well grounded to do bronchoscopic work as the otolaryngologist. In the first place, he is accustomed to working in that particular region. Second, he is accustomed to the more delicate manipulations, and to the handling of similar instruments, as well as to the methods of illumination used in tube work. However, the fact that a man is a practical otolaryngologist does not necessarily mean that he can become a good bronchoscopist, for many reasons. Some men have not the natural knack, and I know of no specialty in which knack is so necessary and important as in this, because such great harm can be done by unskilled manipulation of these instruments. For that reason, I feel that the mere fact that one has done otolaryngologic work is not sufficient justification for his going into this specialty within a specialty. I do feel, however, that the man who takes up bronchoscopic work should really be an otolaryngologist. The development of team work and the necessary skill and the keeping up of that skill that comes thru practice, is impossible in small communities for more than a few. Only two or three men in a given locality can get enough work in this line to keep up their skill, and even tho they practice faithfully to acquire that skill, they cannot have the clinical experience necessary for good work and balanced judgment. Consequently, I feel that altho these otolaryngologic accidents that Dr. Tucker has called to our attention are instructive and interesting, they do not necessarily point to the fact that the otolaryngologist should be prepared to deal with such accidents. In fact, if that were true, then the dentist should be doing bronchoscopies, because there are more such dental accidents than otolaryngologic. I do feel that a well trained bronchoscopist should be in every community of any size, and that this man should be an otolaryngologist primarily; also that he should be supported by all the otolaryngologists. He should limit himself to laryngeal surgery and bronchoscopy if he can, but if the community is not large enough for him to make a living in such a limited specialty, he should still be supported by the otolaryngologists of the community, instead of every man equipping himself with a set of expensive instruments that he will probably use only once or twice a year.

Dr. E. E. Hinman, Albany, N. Y.: Dr. Tucker has called attention to two classes of cases in our otolaryngologic practice—foreign bodies and cases of abscessed conditions developing in the lung. As to the foreign body proposition, there are several points we should bear in mind when doing operative work within the nose and throat, particularly in the oral cavity. First, and this comes to me with particular force, because one of the cases which Dr. Tucker has reported occurred in my practice, in a way that I had never known might happen, but which I will guard

against in the future—the direct examination of the mouth and chest with an X-ray study in any postoperative case in which there is cough. In the case to which I refer, there was loss of a deciduous crown following a tonsillectomy under ether, in a boy of 11 years, which tooth I was quite certain was in his mouth when he left the operating table, but which he had inspirated between that and the next day, because his cough did not begin until twenty-four hours after the operation, and he was out of his anesthetic within a half-hour of leaving the operating room. If there had been a foreign body at the time of operation, I am sure we would have had a reflex cough before twenty-four hours. A direct examination of the chest was made by a very skillful internist, but because the child had had a previous bronchitis, he naturally overlooked the tooth until we later secured the X-ray data. I believe we should all be familiar with what is shown in the skiagrams, and not leave it entirely to the X-ray expert to interpret these skiagrams, because in the average community he does not get enough of them to make a diagnosis. In the case to which I refer, a diagnosis of foreign body was not made until it had been studied thru the skiagram.

You will have noticed that the pictures shown include antero-posterior and lateral skiagrams, and that should be insisted upon wherever you have an X-ray made for the purpose of diagnosis in these foreign body cases, because you may catch it in one and not in another. These cases have a great deal of variation.

I might mention in passing the absence of high temperature in case of metallic substances in the bronchus, as opposed to the immediate temperature response in cases of a vegetable or protein nature.

The important points are the preliminary examination of the mouth for any lost teeth that may have become loosened, the careful application of mouth gags, the necessity of developing an endoscopic technic to at least a moderate degree, and then the prompt reference of all cases that become suspicious to some one who is expert in caring for them, before they get to the point of being entirely lost in the granulations that so promptly develop about a foreign body.

Dr. Thomas J. Gallaher, Denver: I wish to say a few words in regard to what we have a right to expect from the laryngologists in the larger tuberculosis sanitoria. The local treatment of pulmonary conditions, including bronchiectasis, abscess of the lung, etc., in which injection of lipiodol are used, has advanced to such a stage that it appears to be encumbent upon the attending laryngologists in these institutions to carry out this work, and I feel that they should perfect themselves in these procedures. It is certainly a very meritorious accomplishment for any laryngologist to become expert in bronchoscopy and esophagoscopy, and very often certain procedures can be instituted which are of real value to the surgeon. An example is the operation for esophageal diverticulum by the Jackson-Gaub method. Where it is absolutely necessary to do a tracheotomy under general anesthesia, where the patient has much obstruction, this indeed should be done only by passing the trachesoscope and administering the anesthetic thru it; then the operator may cut directly down upon the trachea; however, as pointed out by Jackson, it is better in all such cases to do the tracheotomy under local anesthesia, as sudden death may ensue as a result of abolishing voluntary breathing.

HERPES ZOSTER OTICUS.

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The acute infectious disease known variously as herpes zoster, zona, or more frequently to the laity as shingles, has come down to us from the ancients. Comments in old literature as well as its nomenclature lends to that thought, for the "Zoster" of the Greek signified a girdle, "Zona" or "Cingulum" of the Latin meant a belt or girdle, while an old French dialectic form "Chengle," a girth, passed with its original meaning into the English "Shingles," which remains with us today as the popular name for this affection.

The name herpes zoster, as applied to herpes auris or herpes auriculae, is a misnomer from the standpoint of its usual significance, which is so well exemplified in intercostal herpes; however, it has gradually assumed its place as a proper name for the vesicular eruption of the cephalic extremity which is due to acute infection of the cranial ganglia having representation in this area.

FREQUENCY OF APPEARANCE.

Knowles found 286 cases of general herpes zoster among 31,337 patients with skin affections. Max Joseph, in his study of general herpes zoster, assigns to it the proportion of about one per cent of all skin diseases. Reports from various clinics at home and abroad vary between one and two per cent—a little more than one per cent, as in Knowles' records, being the rule.

Hewlett's statistics place seventy-six per cent of all cases of herpes zoster as affections of the trunk. Greenough's series indicate that herpes zoster of the intercostal nerves occurs fourteen times as frequently as facial herpes zoster. No records giving exact figures on this subject have come to notice, but if a mean is reached between the tabulations of these observers, it is found, approximating roughly, that

herpes zoster of the cephalic extremity makes up from eight to fifteen per cent of all cases of this disease.

If this proportion is even relatively correct, its occurrence in this region is much more frequent than the number of such cases coming under observation of the otolaryngologist would suggest.

Personal inquiry reveals the apparent rarity of the affection in the practice of otologists, some of whom report the observance of a single case or, rarely, two or three, while very many have seen none. In comparison with other aural affections, case reports, discussions or studies of herpes zoster affecting the ear and contiguous parts are relatively infrequent in the literature.

Ramsay Hunt[1], in 1907, in order to determine the relative frequency of the occurrence of herpes zoster oticus, examined the records of a number of hospitals with the following results: At the Manhattan Eye and Ear Hospital, covering a period of ten years and 47,600 cases, the diagnosis of herpes of the auricle was made twice. In the Brooklyn Eye and Ear Hospital, with a total of 15,000 cases in five years, the diagnosis was made once. The New York Eye and Ear Infirmary, with an average of 10,000 out-patients yearly, recorded this diagnosis six times. At the Massachusetts Eye and Ear Infirmary, in the preceding period of ten years, covering 65,000 patients, this diagnosis was made thirty-three times.

The same author[2], in 1910, after an exhaustive search of the literature, reported a series of cases of herpes zoster of the cephalic extremity to the number of eighty-seven, twenty-seven of these being cases of pure herpes zoster oticus. In 1915, he[3] gave out an additional series of twenty cases of herpes zoster oticus. This series represents probably the majority of all such cases reported to that date, and gives a fairly accurate idea of the relative frequency of diagnosed and reported cases.

The scope of this paper is not limited to herpes zoster oticus, but includes as well all cases in which any portion of the external ear is affected by the vesicular eruption of herpes zoster. This brings into the picture the sensory aural distribution of the Gasserian ganglion and of the upper cervical ganglia, and thereby increases materially the number of cases which may be properly considered in the field of the otologist.

Vail[4] in 1906, Dabney[5] in 1914, Beck[6] in 1914, J. E. Brown[7] in 1917, F. P. Emerson[8], F. E. Dennis[9] and J. A. Watson[10] within the past four years—all members of this society, or of the Triological, have reported cases and made valuable contributions to the literature of this subject. To J. Ramsey Hunt, however, belongs the honor of having placed herpes zoster of the cephalic extremity on the plane of a definite clinical entity. His monumental work in developing all phases of the subject is comparable to that of Head and Campbell in establishing the pathology of general herpes zoster.

Neurology.

Very brief mention will be made of the intricate and complex sensory innervation of the ear. The nerves taking part in this innervation are the fifth, seventh, eighth, ninth and tenth cranial nerves, and the great auricular and small occipital nerves from the cervical plexus.

The ganglionic representation of the ear is placed in the Gasserian, geniculate, petrous, jugular and the second and third cervical ganglia. The nerve supply of the auricle, external auditory meatus and canal, and the tympanic membrane have had many careful studies. Cushing's[11] study of the auricular fields of anesthesia after extirpation of the Gasserian ganglion and the upper cervical ganglia, is outstanding in its completeness and thoroness.

The accompanying illustrations, taken from his work, show clearly not only the sensory limitations on the external ear of the auriculotemporal branch of the fifth nerve anteriorly, and the great auricular and small occipital nerves posteriorally, but also a definite intervening area of esthesia between these anterior and posterior limits. Hunt, by the zosterian method of study as well as by association of related studies in comparative anatomy and embryology, has assigned neural representation of this area to the petrous, jugular and geniculate ganglia.

The very frequent association of a seventh nerve paralysis in this syndrome, makes necessary mention of the accepted fact that the facial is a mixed nerve; the geniculate ganglion, placed on the nerve just within the entrance of the aqueductus Fallopii, is homologous to the posterior root or sensory ganglia of the spinal nerves, and similar in type to the Gasserian, jugular and petrous ganglia, and there seems no doubt

that all of these cranial ganglia are just as susceptible to the virus producing herpes zoster as are the spinal ganglia.

These ganglia have also an intraoral sensory representation, and a unilateral vesicular eruption affecting the tongue, fauces, and pharynx has been noted alone as well as in association with auricular or facial herpes zoster.

Pathology.

The underlying pathology of herpes zoster was for a long period conjectural. The first to suggest that the eruption follows the distribution of the nerves was Mehlis in 1818. In 1861, von Barensprung[12] reported the first autopsy, with findings in which he demonstrated clearly involvements of the posterior root ganglia.

Wyss, in 1871, and Sattler, in 1875, reported postmortem findings in a case of herpes zoster involving the ophthalmic or first division of the trigeminal nerve. In each case, the Gasserian ganglion was found to be the seat of an inflammatory lesion. It was not, however, until 1900 that Head and Campbell[13] gave out the results of their exhaustive clinical and pathologic study of herpes zoster, which included the pathologic material obtained from twenty-one autopsies and placed this part of the subject on a substantial basis. These findings may be summarized briefly as a hemorrhagic inflammation of the posterior root ganglia, degeneration of the posterior root nerves and of the peripheral nerves as well as the root fibers in the posterior column of the spinal cord —secondary changes following according to the severity, extent and duration of the disease. The autopsies followed the onset of the affection from a few days to one and one-half years.

General herpes zoster affects usually a single ganglion, and the efflorescence is found along the cutaneous distribution of the corresponding peripheral nerve. Curious anomalies and variations from this rule have been observed; two or more ganglia may be affected at the same time, giving rise to a corresponding extension of the herpetic zone. Altho the infection is usually limited to one side, and this sharp delimitation is one of its strong diagnostic points, cases of bilateral involvement have been reported. This anomaly is still more unusual when the bilateral affection arises at different levels of the spinal segments. Unusual, also, is a homolateral in-

volvement of widely separated ganglia, of which cases have been published.

The association of paralysis of the corresponding motor nerve in the areas covered by the spinal ganglia is rather rare; much less so is this the case in zoster of the cephalic extremity, where a seventh nerve palsy is rather frequent. Exceptional, indeed, is a facial paralysis complicating zoster of an intercostal nerve, but it has been observed.

Etiology.

The etiology of this disease has not been finally determined. Rivers and Tillett[14] have made up a table suggesting a relationship between sheep, horses, and cow pox, small pox, varioloid, alastrim, chicken pox and herpes zoster, symptomatic herpes and epidemic encephalitis; all of which, except the latter, have in common a characteristic vesicular eruption of the skin which shows similar histologic characteristics in most of them. A large literature on the similarity of herpes simplex, varicella and herpes zoster, and the probability of an identical etiology has been developed. More recently, epidemic encephalitis has been included in this group, on the ground of its origin from the same or a similar virus. In none of these conditions has the etiologic agent been isolated or cultivated, but the available evidence points to an ultramicroscopic or filtrable virus as the causal factor.

Cole and Kuttner[15] report as unsuccessful a series of experiments to inoculate rabbits, guinea pigs and monkeys from 9 cases of herpes zoster. They discuss the experimental work of Teague and Goodpasture, Lipschutz, Kundratitz and a number of others, and conclude that until herpes zoster can be regularly transmitted to animals and cross immunity test carried out, the relation of the virus of herpes zoster to that of herpes simplex remains a matter of speculation, and it does not seem likely that the etiologic agents can be identical.

The question of the identity of herpes zoster and varicella is still more difficult to answer, because neither disease at present is easily transmissible to animals. Kundratitz' observations indicate a close immuniologic relationship between herpes zoster and varicella, and seem to show the presence of a transmissible virus in the vesicles of herpes zoster. Thalhimer[16] recently reported a fatal case of typical cervical herpes zoster, which at autopsy showed histologic findings in the central nervous system quite similar to epidemic encephalitis.

Aside from the specific agent which invades the neural ganglia, there must be other factors at work to induce or predispose to the disease. Some of these cases follow exposure to the elements, with chilling of the body, or appear after walking or driving in the face of a frigid wind. A number of cases follow or are associated with attacks of influenza. In others are found chronic nervous diseases, such as general paralysis or tabes. Head colds and sore throat are likewise mentioned as associated.

The association of head colds and influenzal attacks brings to mind the nasal sinuses and accessory cells, with the thought of their possible relationship. The possibility of the branches and endings of the peripheral nerves traversing the nasal mucosa and that of the paranasal cavities being concerned in the direct transmission of infection to the Gasserian ganglion, has been suggested. With the same thought in mind, one may add the peripheral nerve distribution in the mucosa of the buccal, pharyngeal and laryngeal cavities, as possible conductors of an infective agent to the various ganglia having to do with herpes zoster of the cephalic extremity. Naturally, infective disease of the nasal cavities, tonsils or teeth must be considered as a possible factor in favoring the onset

The presence of vesicular eruption, in so far as the writer knows, has not been demonstrated in the mucosa of the middle ear or of the paranasal cells; but in the buccal, pharyngeal and laryngeal cavities, herpes strictly limited to the lateral half of these parts and conforming in other respects to cutaneous zoster, has been identified and recorded a number of times. One may recall Teague and Goodpasture's hypothesis, that the virus of herpes first multiplies at the site of inoculation in the skin and passes up the corresponding spinal nerve to its spinal ganglion; the virus then seems to pass centrifugally along the nerve and its branches to the skin where it multiplies rapidly and gives rise to characteristic herpetic vesicles. Should this hypothesis prove to be correct, it seems probable that in many cases transfer of the specific virus to one or more of the cranial ganglia may be along the nerve paths in the mucosa noted above.

Symptomatology: Prodromal Symptoms.

The attack may be ushered in by mild prodromal symptoms, with a feeling of malaise for a few days, followed by slight temperature and a feeling of general discomfort or

aching; or the prodromata may be more severe, with a feeling of general illness and severe headache, succeeded sometimes by a violent chill with nausea and vomiting (case 2), or there may be no marked initial symptoms, the disease arising suddenly with or without a chill, violent pain being the first symptom to appear.

Sensory Symptoms.

If the geniculate ganglion is affected alone, or is the chief point of attack, the first symptom is usually a severe pain in the ear, lancinating, throbbing or deep aching in type. This preherpetic pain may reach a high degree of intensity, radiating perhaps to adjacent areas and prostrating the patient by its violence. If the upper cervical ganglia are chiefly affected, the first sensory symptom may be a sense of burning discomfort over the neck and occiput, which later develops into pain of the types noted above. Should the Gasserian ganglion be attacked, the same sequence may be noted on the face, or less frequently, perhaps, the last two types may have their sensory onset with characteristic pain. The pain usually gives way after the appearance of the eruption, but may persist as a postherpetic pain for many months, and this may take the form of a violent otalgia or mastoidalgia. Especially in the aged, sensory symptoms of burning, discomfort, pain or paresthesia may persist in the affected areas for many months or longer (in one of the author's cases still present after one and one-half years). These sensory symptoms may be more annoying at night, causing loss of sleep with its attendant ills.

Hypesthesia, or obtunded sensation, has been noted in the concha and external auditory meatus in recent cases of herpes zoster oticus with facial paralysis. In one of the author's patients, a case of facial and auricular herpes zoster with facial paralysis, a decided hypesthesia is still to be noted in the concha, external auditory meatus and nearby points, after the lapse of one and one-half years.

At the onset of the disease, rarely has pain affected one side of the throat, giving rise to added discomfort and pain when swallowing, radiating then to the ear or to the mastoid or both. The reported cases developed herpetic eruptions over the intraoral distribution of the glossopharyngeal, vagus or facial nerves. Pain and soreness along the edge of

the tongue have been noted rarely (case 1). It is to be noted that herpes zoster has affected the innervations mentioned above without giving pain.

The Vesicular Eruption and Accompanying Inflammatory Reaction.

Within a few days of the definite onset of the disease, a vesicular eruption appears. In the author's cases, the eruption was preceded by a diffuse redness of the part and a definite infiltration or swelling of the underlying tissues, forming, so to speak, an inflammatory base upon which the vesicles arose. This has been recorded in the case of other patients, so that it is, no doubt, frequently the first objective sign to be noted. If the eruption is limited to the external ear alone, its distribution will be found in the zoster zones of the geniculate, petrous and jugular ganglia, which, as outlined by Hunt, occupy a cone shaped area from the posterior part of the tympanic membrane along the posterior wall of the external auditory meatus, include the concha, tragus, antitragus, incisura antitragica, antihelix and fossa of the antihelix, the upper part of the external surface of the lobule, and occasionally a small area on the postero-mesial surface of the auricle and adjacent mastoid. If other parts of the auricle are affected, the Gasserian ganglion in part or the upper cervical ganglia or both must be affected, and a facial or occipitocollaric eruption alone or combined will be associated with the auricular efflorescence.

The vesicles may appear in small or large groups, scattered or almost confluent, and may occur in crops, a day or two or more between successive crops. In isolated herpes zoster oticus, there may be out two or three vesicles in the concha or in the external auditory meatus or rarely one or two or more have been observed on the tympanic membrane (Dabney's[5] case). In other cases, a considerable number of vesicles appear in the zoster zone of the geniculate genglion. These likewise may appear in successive crops. The skin of the ear may become red and swollen and the entire auricle take on an erysipelatous appearance.

In cases of the latter type, the soft parts of the external auditory canal may become swollen, until it is difficult to examine the tympanic membrane and its landmarks. Should the vesicles be numerous and contain a considerable amount

of serum, the rupture and drainage of several crops of these may, with other symptoms, give every indication of a middle ear inflammation which is draining thru a spontaneous rupture of the tympanic membrane.

Usually, after a few days the vesicles desiccate and the soft parts begin to lose their swelling and edema; in from two to four weeks, the appearance of the ear is much the same as before with the exception of remaining herpetic spots. These spots vary in appearance from small pigmented areas to cicatrices, which mark a healed ulcer in the deeper layers of the skin. Such postherpetic scars limited unilaterally and to the peripheral distribution of a cutaneous nerve as well, may be sufficient evidence from which to elicit a history of the primary attack.

A small group of cases has been recorded in which an herpetic eruption was observed within the oral cavity. The vesicles in these cases were limited unilaterally as well as to a definite neural distribution, in contradistinction to the large number of cases in which vesicles of a different character are seen on bilateral areas of the buccal mucosa, and are not to be considered as manifestation of idiopathic herpes zoster.

This intraoral appearance has been noted on the tongue, the soft and hard palate, the uvula, the faucial pillars and tonsils, the pharynx, the epiglottis, the arytenoid cartilages and aryepiglottidean folds. The eruption in this type of case, appearing as it does upon mucous surfaces, is more evanescent, gives way more rapidly under heat and moisture of the mouth and leaves whitish or yellowish flecks, quite unlike our usual conception of a vesicular eruption. (Hunt.)

Paralytic Complications.

Facial paralysis complicating herpes zoster of the cephalic extremity is apparently very frequent, for Ramsey Hunt, in 1910[2], reported a series of eighty-seven cases of zoster of this region, all of which were accompanied by seventh nerve palsy. In 1915[3], the same author refers to an additional group of cases of herpes zoster oticus, twenty in number, all of which had an associated paralysis of the facial nerve. This paralysis usually involves all three branches and is complete.

Since this nerve supplies all the facial muscles from the occipitalis to the platysma, the onset of paralysis with its

cardinal symptoms of staring eye, flat face and twisted mouth, with lacrimal secretion overflowing and saliva dribbling from the angle of the mouth, is often quite alarming to the family, who feel, despite your assurance to the contrary, that it is the beginning of a stroke of paralysis. It is not unusual for the chorda tympani to show involvement by loss of the sense of taste in its distribution.

Paralysis of the soft palate, evidenced by drooping of the affected palatal arch and, on innervation, by elevation of the opposite arch and towards the nonaffected side, has been observed a number of times. That this palsy may result from inflammatory reaction in the glossopharyngeal or vagal ganglia has been suggested (Hunt). Laryngeal paralysis in the course of herpes zoster of the cephalic extremity has been observed (Dan Mackenzie[17]).

Rarely, paralysis of one of the motor nerves of the eye has been recorded as a complication of herpes zoster; out of Hunt's series of eighty-seven cases of herpes zoster of the cephalic extremity with paralysis of the facial nerve or auditory symptoms, but one case had an associated paralysis of a homolateral ocular nerve. Case No. 1, here recorded, is another of the same type.

One point of especial note is the evanescence of these palsies of the face, palate and larynx, and also of the cochlear and vestibular nerves, if we include them in paralytic complications. Frequently, marked improvement in one or more or all of them may be observed within a week or two of the onset. Middle class cases, which entirely clear in the course of many months, are also to be found. Rarely, either a partial or complete paralysis of the face is the final outcome. The limited duration of these palsies probably has somewhat to do with the less frequent observation and report of the palatal, and especially the laryngeal form.

Hearing.

The state of audition may vary from hyperacusis with various gradation to total deafness. Hearing may be affected thru closure of the external canal by inflammatory reaction and swelling of the soft parts. In these cases, Weber will lateralize to the affected side, unless a well marked perceptive deafness had been present in this ear, prior to the attack of zoster. Again, hearing may be dulled when desquama-

tive debris has partly filled the canal. With its removal, the dullness will pass. Finally, one may by test uncover a diminution of hearing of perceptive type of which the patient was unaware; this will add to any difficulty in diagnosis.

These types are not of especial interest, save that they must be eliminated in diagnosis in this disease, but those where there is deafness due to specific inflammation of the ganglion of Corti are much more in question. In this type, the auditory acuity varies from hypacusis to total deafness. The frequent occurrence of auditory symptoms in herpes zoster of the cephalic extremity can be judged by the statistics of Hunt, who records twenty-six cases out of eighty-seven as presenting such symptoms in varying gradations.

Vertigo and Disturbance of Equilibrium.

These may or may not be associated with deafness. In a number of reported cases (Dennis[9]), the Meniere symptom complex of tinnitus, nausea and vomiting, deafness, vertigo and disturbance of equilibrium, was an outstanding feature of the disease.

That these cases arise from an acute specific affection of the ganglia of Corti and Scarpa, there seems slight reason to doubt. No mention will be made of the clinical, anatomic and embryologic evidence or the studies in comparative anatomy utilized to buttress this theory, which has not as yet been confirmed by pathologic findings.

While deafness as well as vertigo usually passes away, it is in the type of case just mentioned, that total deafness is most likely to be a permanent symptom. Vertigo and disturbance of equilibrium, even tho severe, gradually pass as compensatory adjustment progresses. In several reported cases, the cochlear and vestibular labyrinth have remained inexcitable to all stimuli, with resulting permanent deafness.

Diagnosis.

Diagnosis today of herpes zoster of the auricle is perhaps not difficult, when all the cardinal symptoms of a well marked case, appear in sequence. But twenty years ago, when Vail wrote his thesis on this subject, he called attention in the first paragraph of his paper to the fact that among the various text books of otology he had consulted, but two, those of Dench and Burnett, gave a thoro description of the disease. The others either passed it by, or, if mentioned at all, dis-

cussed the subject in a paragraph not exceeding fifteen lines in length.

Naturally the undergraduate and postgraduate student of that time had slight opportunity of gaining even a reading acquaintance with the disease, and consequently there was a much greater possibility of overlooking a case, even tho not particularly difficult of diagnosis. The status today is quite different, for recent text books contain clean cut descriptions of the disease. Furthermore, since Hunt's extensive work along this line, there has been occasional case reports and discussions in otologic journals (altho as stated above, these are relatively infrequent), so that no one conversant with current literature need be without at least a slight acquaintance with the subject.

The history of a severe pain in the aural region preceded by the prodromal symptoms mentioned, and followed shortly after by an inflammatory reaction and a vesicular eruption affecting the auricle, the external auditory meatus and canal or tympanic membrane, this accompanied by, or a few days later associated with, a paralysis of the facial nerve of the same side, is pathognomic of the disease. The same symptoms without the advent of seventh nerve palsy, but with the addition of auditory symptoms of either defect of hearing or vertigo and disturbance of equilibrium or both, is equally pathognomonic.

In such clean cut types, it is improbable that an error in diagnosis will be made even tho the otolaryngologist has no other acquaintance with the disease than his reading of Dabney's[5] critical essay of 1914, or that of Brown[7] of 1917, or has taken note of Dennis'[9] thoroly reported case, with his discussion of the outstanding symptomology of the disease, or lastly has come in contact with J. A. Watson's[10] report of two cases with a presentation of some of the cardinal points of the disease. But many of the cases are much more difficult of diagnosis.

When the auricle remains swollen and crusts are to be seen here and there, giving at first glance the appearance of an acute eczema which has arisen as the result of an acute purulent otitis, and this fits in so well with other clinical symptoms, no little diagnostic skill is required to separate the two.

The various forms of myringitis must be considered, and especially those in which large vesicles or bullae are found on

the tympanic membrane and at times also on the wall of the external canal near by. These cases are found especially during or following an influenza attack, and are met with quite frequently. In comparison, herpes zoster oticus is rare, is very often accompanied by a facial paralysis, and in one case out of three by eighth nerve disturbances, while its vesicular distribution is seldom found on the tympanic membrane, but usually appears on the auricle (the central parts in particular) the external auditory meatus and at times the external canal; so that as a rule they will not be difficult to differentiate.

Dabney[5] writes of how Bonnier faced a problem in diagnosis when he encountered a tuberculous syphilitic who exhibited ophthalmic and auricular herpes with vesicles along the temporoauricular distribution and six on the membrana tympani. In the light of the protean manifestations of lues, this picture would baffle many for a time, if not for all time. In some of the cases, the symptoms closely simulate that of acute suppuration of the middle ear with associated mastoid pain and perhaps tenderness, or enlargement of the cervical glands. When we add to this paralysis of the cranial nerves, including the abducens (in one of the author's cases), despite the fact that the radiogram is negative, there may still be a suspicion that the disease is deeply seated in the cells of the petrous bone. In such a case, if the vesicular distribution had been small and had not been observed particularly by the family or the internist, it would seem hardly possible for the otologist to make a correct diagnosis, and that he would undertake surgical intervention in some form seems positive.

Kaufmann[18] reports a case in which the diagnosis of acute mastoiditis was made and a mastoidectomy done, and Halphen[19] describes a case that seemed to be one of grave mastoid disease, for it appeared to present acute otitis media, acute mastoiditis, deafness, and possible thrombosis of the lateral sinus, all due to the great mastoid pain, inflamed drum, swelling of the neck and facial palsy.

When confronted with cases presenting such serious symptoms, and a clean cut history of a regional herpes zoster cannot be obtained, the radiogram will establish frequently the condition of the mastoid and its cellular spaces. An examination of the spinal fluid may also be of value, first in determining an increased pressure, and second as to its cell con-

tent. The blood picture in certain cases may give some points toward diagnosis.

From a study of the literature and an analysis of reported cases as well as those personally observed, the writer is led to believe that in many instances the tympanic membrane has been incised, and at other times mastoid surgery undertaken, in cases where this disease was not recognized; that mistakes in diagnosis may arise because many of these patients do not come under observation of an otolaryngologist, or because the essential elements for correct diagnosis could not be or were not elicited in the history and were not present for observation when the patient came under the care of an aural surgeon; that herpes zoster of the cephalic extremity is less rare than it seems, and that the prompt investigation of all the symptoms associated with the relatively numerous facial palsies would place some of them at least as dependent upon affection of the geniculate ganglion by the specific virus of this disease.

Case Reports.

Case 1. March 4, 1925, N. P., 70 years of age, a stonemason by occupation, was referred to me by his physician with a diagnosis of acute mastoiditis, and with the suggestion of the necessity for immediate operation in view of the alarming symptoms he had exhibited for the preceding two weeks.

Mr. P. has always had rugged health, never having spent a day in bed on account of illness, to his recollection.

Four weeks ago, he had an attack of grippe with fever, aching and soreness in the bones and muscles, especially soreness and stiffness of the neck muscles on the right. He was confined to the house but not to his bed thru this attack. Fifteen days ago, he was taken with pain affecting the right side of his face, head, ear and mastoid and radiating into the neck muscles. The same night, about 10 o'clock, he had a chill, and two hours later the pain thru the areas noted became agonizing. The following morning, the right auricle was red and becoming swollen. At the same time, vesicles were noted on the face in front of and above the level of the ear. Two or three days later, vesicles appeared in the central parts of the auricle in the external auditory meatus. The same day, another crop of vesicles appeared on the face in front of the ear and over the angle of the jaw. The auricle by this time was swollen and quite erysipelatous in appear-

ance. Two or three days after their appearance, the first vesicles had begun to desiccate, and two or three days later, those within the cavity of the auricle and external auditory meatus ruptured and a sanguinous serum in small amounts came from this region.

About the same time that the vesicular eruption appeared within the auricle, the right side of his tongue became much inflamed and swollen, and was covered with vesicles limited strictly to the same side. The vesicles on the tongue healed about as rapidly as those on the face. There were no vesicles on the buccal mucosa, but his lips on their right side were much inflamed. Two weeks after the onset of the disease, paralysis of the right side of the face was noted, and at the same time an internal squint of the right eye was observed by the family. There was no pain in the throat, or pain or soreness in swallowing food. Normal taste of food, however, was lacking.

With the onset of the vesicular eruption on the face and the coincident swelling of the auricle, he suffered from an intense vertigo and was unable to stand alone, falling to the right side. This vertigo has greatly decreased but he still takes the arm of a companion in walking or places his hand against the wall or object of furniture when moving about the house unassisted.

His hearing was also affected, with the association of well marked tinnitus. His hearing has improved but he thinks it is still much below normal.

Examination. Temperature, pulse and respiration are normal.

Strong, vigorous looking man of 70, who has the appearance of having suffered extreme pain. The musculature in the distribution of the facial nerve and the motor branch of the trigeminal nerve on the left side is normal in action. On the right side, however, the face is flat and forehead cannot be wrinkled, but there seems to be no impairment in the muscles of mastication.

Eyes. There is a convergent squint of the right eye. The eyeball cannot be rotated lateralward beyond the midline, indicating a complete paralysis of the sixth nerve. Other external and internal nerves of the eye seem unaffected, and there is no disturbance of any of the ocular nerves on the left side. The pupils are 2 mm. in size, react to light, con-

vergence and consensually. The vision with each eye with correcting lenses is 20/20 minus.

Nose. The nasal cavities are negative.

Mouth. The lips, the tongue, the mucosal covering, the fauces, uvula and pharynx, are somewhat inflamed on the right side. All of his teeth have been extracted. The tongue protrudes straight. The tonsils are submerged but are of moderate size.

Ear. The left ear shows nothing abnormal. The right auricle is still somewhat red and inflamed. The region of the concha and external meatus is covered with softened debris, which, when removed, has a slightly fetid odor. A desquamative dermatitis is present in the canal, which, when gently removed, leaves an inflamed base. The tympanic membrane is covered by desquamating epithelium. There is no bulging, and the membrane and ossicles move readily with the pneumatic otoscope. No evidence of perforation of the tympanic membrane is seen.

Hearing. Left ear = conversational voice, 40 ft. plus; whisper, 9 inches.

Right ear = conversational voice, 40 ft.; whisper, 6 inches.

Station: With eyes closed, sways markedly to the right and finally falls. Direction of falling varies with change of position of the head.

Gait. With eyes closed, walks to the right and falls if not supported. Patellar reflexes present.

Three weeks later, the patient's symptoms were somewhat improved. Within three months, he was able to open and close the right eye perfectly, the ocular rotations were normal in the field of the paralyzed muscle, and movement of the face had much improved.

After six months, he had no symptoms that gave him real trouble except paresthesia, soreness and pain in the facial region which the vesicular eruption had affected, and also a sensation of burning along the right side of the tongue and of the lips. Following the appearance of the facial paralysis, the pain in the ear had lessened rapidly, and at this time simply an occasional pain in the ear and mastoid were noted.

A recent examination of this patient shows a strong, rugged looking man, 72 years of age, the forehead wrinkled, and the eyelids open and close normally. He is just regaining the power to whistle for his dog. Sensation is the same on

both sides of the face. He protrudes the tongue straight and sensation is the same on both sides. There is no sign of muscular weakness in the palatal musculature.

Eyes. Pupils are small, 2 mm. react to light, convergence and consensually. Vision with correcting lenses is a little less than 20/20 minus in each eye.

Ears. Show nothing abnormal. The hearing for whisper is three inches for right ear and nine inches for the left ear, with Weber lateralizing to the right. Sensation to touch in the concha and auditory canal of the right ear is decidedly obtunded. His station is good and reflexes active.

He states that paresthesia, soreness and pain in the face is as severe as ever, and so extremely annoying at night as to seriously disturb his sleep. He mentions, also, occasional pains in the ear and mastoid, which are neither frequent nor severe.

This case is of unusual interest for several reasons:

1. The coincident involvement in sequence of six cranial nerves, from the fifth to the tenth, with ganglionic relation to the Gasserian, geniculate, auditory, petrous and jugular ganglia. This illustrates admirably the multiple involvement of ganglia in herpes zoster of the cephalic extremity.

2. The association of an intraoral herpetic affection, zosterian in type, of which but few cases have been recorded.

3. The presence of a sixth nerve paralysis complicating herpes zoster of the face, ear and tongue. Abducens paralysis has been reported a few times in association with herpes zoster ophthalmicus; but Hunt, in an exhaustive search of the literature, found but one case of herpes zoster of the cephalic extremity complicated by facial palsy and auditory symptoms with which an ocular nerve paralysis was combined, and in this case the sixth nerve was affected.

The causal factors influencing this sixth nerve palsy cannot be determined, but in attempting to account for its presence one may discuss certain features. It is of course peripheral in character, that is to say an affection of the nerve fibers at some point between the nucleus and the ending of the nerve in the external rectus muscle. Affection of the nerve at any point within the orbit seems improbable for the reason that sixth nerve palsy is recorded so rarely in herpes zoster ophthalmicus, in which the tissues about the orbit are always affected, and even the globe and other intraorbital tissues

frequently may be. That it is the result of pressure from an increased volume of cerebrospinal fluid (this increase seems not uncommon) also seems unlikely, for the acute period of the disease had passed before the palsy appeared, and there were no other symptoms of such pressure. That it was a toxic paralysis in the correct meaning of the term, such as may occur in diphtheria, for example, is not probable, for the infective agent inducing herpes zoster has a selective affinity for the unipolar cells of the sensory ganglia and not for the nerve primarily.

Aynaud's[20] case of abducens palsy complicating facial and auricular herpes zoster with facial paralysis and auditory symptoms (cited by Hunt) is the only one noted by the writer in a review of the literature. This case had an associated intraoral herpes as had the case under discussion. The fact that both of these cases had an herpetic involvement of the mucosa within the oral and pharyngeal cavities, leads to the thought that concurrently a vesicular inflammation affecting the mucous membrane of the sphenoid sinus and ethmoid cells may have developed.

The work of Sluder and Onodi[21] has shown how frequently the sphenoid cavity is of large size, with its walls thin as paper and presenting dehiscences; their work has shown also the frequency of an intimate relationship between the sixth nerve and the contiguous sphenoidal wall. An affection of this nerve from inflammatory reaction within the sphenoid sinus is on record.

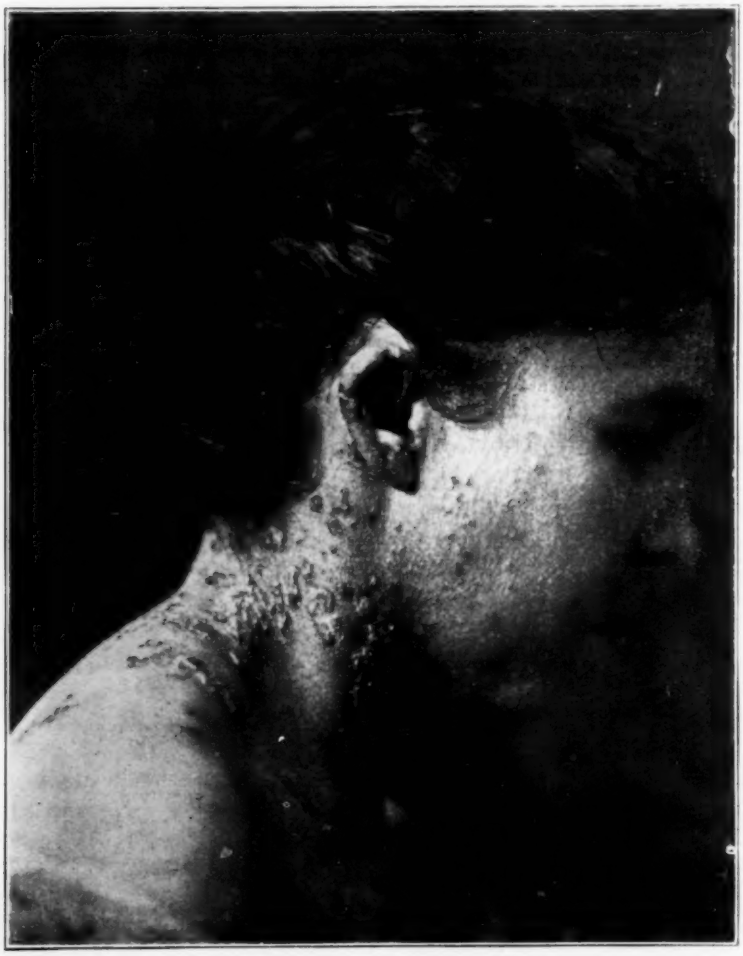
That the pathology outlined was causal in the production of the abducen paralysis in both of these cases seems not only possible but more likely than the other methods discussed, and is here suggested as the probable cause in the author's case.

Case 2. Miss E. L. P. M. H. Has had the usual diseases of children, but no serious disease of adult life. Her health has usually been fairly good and she passed thru the three years of nurse's training without difficulty. April last, she had a slight attack of hyperthyroidism; this was of short duration, and since then she has been perfectly well, altho during the week of July 4th she had at times a hemiopia of the right visual field lasting for a few minutes.

July 15th, she developed an intense pain in the top of the head, thru the forehead and into the eyes. The next morning

the headache was gone, but her head was sore and the eyes particularly sore on movement. On the following day, she returned home fearing the onset of an acute illness.

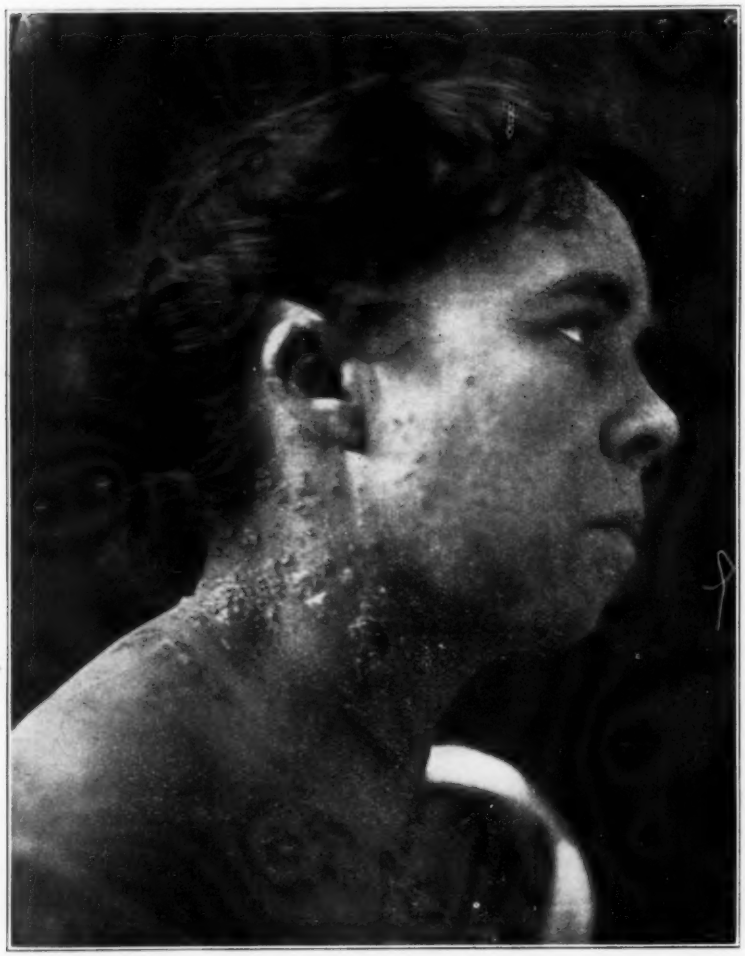
July 18th, she felt rather ill and toward evening went to bed; shortly afterward she had an intense chill lasting for ten or fifteen minutes. Chilly sensations persisted at intervals for several days, intense nausea with vomiting following the



Photograph taken one week after the appearance of the eruption of the ear. The vesicles have desiccated and are disappearing rapidly, altho small herpetic spots can still be seen in the concha, on the antitragus and lobule. The swollen appearance of the helix, with drying crusts and small vesicles, can be noted. The suprascapular eruption, with the sharp delimitation of vesicular process at the midline of the nuchal region, is well shown.

chill. July 19th, she felt better but had a temperature of 102, so returned to bed and remained for the greater part of two weeks. Her temperature persisted for eight days, returning to normal on the 9th.

During the 19th, there was a feeling of paresthesia and burning discomfort over the right neck, an inflamed and reddened surface appearing in this region. The following day,



In this view is brought out especially the sharp delimitation of the vesicular process at the midline of the chest and neck. On the mastoid can be seen drying crusts and vesicles, and these extended to the posteromesial surface of the auricle and adjacent mastoid, the area supplied by the auricular branch of the vagus and the facial nerves. The occipitocollaric eruption, with its sharp unilateral limitation at the midline anteriorly and posteriorly, is of itself diagnostic.

a vesicular eruption appeared in the right supra- and infraclavicular regions, and posteriorly from the deltoid region to the midline of the neck.

After a second interval of two days, the right auricle became inflamed and swollen, and vesicles appeared on the helix from the root over a considerable part of its extent. There were a few small vesicles in the concha, on the antitragus, in the incisura intertragica, and on the outer surface of the lobe of the ear. There were also a few vesicles scattered over the mastoid, and a small group appeared on the posteromesial surface of the auricle and adjacent mastoid, corresponding to the distribution of the vagus in that region. There were no vesicles in the external canal or on the tympanic membrane.

Coincident with the eruption on the ear, vesicles appeared over the face as high as the inferior distribution of the auriculotemporal nerve. All of the vesicles on the face, the mastoid, and the auricle were much smaller than those of the occipitocollaric region.

The photograph of this case was made one week after the appearance of the eruption, when it was already regressing markedly over the aural and facial regions.

It has been noted before, that the eruption on the neck was preceded by a feeling of burning discomfort and paresthesia. This was true likewise of the face and ear. However, in both cases these sensory symptoms changed to soreness and then pain of the lancinating character, which was at times excruciating. The pain of the ear was limited largely to its depths, and this pain and that in the mastoid were the last to make their appearance. With the onset of the eruption, the pain lessened materially, altho two weeks later there were not infrequent pains in the occipital region and over the shoulder, (these pains had extended originally down the right arm to the wrist). At that time she had also had occasional ear pains, but not of great severity.

The acuity of hearing was somewhat lessened, there has been also some feeling of dizziness especially upon sitting up in bed, when she has a sensation of falling to the left and falls unless supported. This feeling is also present when she turns from one side to the other. The sense of taste has been disturbed, first in a partial loss of taste, and second in that there is a constant metallic taste in the mouth somewhat similar to that of iodin.

Examination. A well nourished young woman, the facial muscles normally active on both sides. The eyes are negative. On the right side of the face, external ear, mastoid, neck, supra- and infraclavicular and the suprascapular regions, a vesicular eruption of herpes zoster as described above is present.

Hearing. A test of hearing for voice and watch gave the following: Right ear whisper at twelve ft. and watch at one ft., left ear whisper at forty ft. and watch at four ft. Weber indifferent.

Vertigo and Equilibrium. Standing with eyes closed, sways to the left. Walking with eyes closed, walks to the left.

Mouth. There is no inflammatory reaction in the mouth or pharynx, but there is a slight drooping of the right palatal arch; on innervation, the left arch is more active in its response. Taste has already been noted. The symptoms narrated above passed away gradually. Within a few weeks the vesicles and crusts disappeared; hearing returned to normal; vertigo cleared and the sense of taste returned.

Five months after the onset of the attack, the only symptom left was a feeling of soreness or tenderness about the neck and shoulder and a few scars in this region.

COMMENT. In this case, the chief point of attack was in the third and fourth cervical ganglia, the occipitocollaric eruption corresponding closely to Head's segmental outlines. The severe pains along the nerves of the brachial plexus, altho no affection of the skin was in evidence, indicates at least a mild affection in that direction.

The vesicular eruption on the face, ear and mastoid, less marked than that of the neck, points to a shading off in the severity of the infection from the second cervical to the Gasserian ganglion. Loss of the normal with perversion of the sense of taste, the presence of definite altho mild auditory symptoms, a paresis of the homolateral arch of the palate, as well as the presence of vesicles in the peripheral zones of the auriculotemporal, facial (geniculate ganglion), and auricular branch of the vagus nerves, combine to make plausible the thought of infection of the entire chain of sensory cranial ganglia having to do with innervation of the parts mentioned.

CASE 3. Mrs. M. B., age 50, occupation, housewife. May 14, referred by Dr. O. C. Mierly on account of left facial paralysis and eruption on the left auricle.

P. M. H.: The patient had the usual diseases of childhood except diphtheria and scarlet fever and has had no serious illness

since then. Her appetite is usually good, she sleeps and rests well and has always been muscularly active.

H. P. I.: On May 3rd (eleven days ago), a pain of severe type affected the left side of the face, mastoid, neck and shoulder; this became more severe until May 4th (eight days ago), when an erruption appeared on the auricle. The following day, a complete paralysis of the left side of the face appeared. At the same time the mouth became dry and a bitter taste was noted. The pain became less severe after onset of the palsy and has gradually disappeared until today, when an occasional twinge affects the face and mastoid. Hearing of the left ear was much lowered in the first few days of the attack, and dizziness of mild degree was noted. The hearing today is better, and there has been no dizziness for three or four days. Slight improvement of the facial paralysis is noted, altho dryness of the oral cavity and bitterness of taste persist unchanged.

Examination. Very well nourished woman. Facial muscles normally active on the right side but paretic on the left. On forced closure of the eyes, the left lids can be closed about one-half. The angle of the mouth falls slightly on the left, and she can not show her teeth or smile on that side. Sensation is normal on both sides.

Eyes. Pupils 3 mm., react freely and equally to light and convergence and consensually; ocular rotations normal; no gross lesions of the fundi; epiphoria present left side.

Nose. Septal deflection to left; no inflammatory reaction to be seen.

Mouth. Tongue protrudes to left tho she can at command push it to the right. Taste is normal for sweet, acid and bitter. Upper teeth removed, lower teeth in very bad condition. Anterior faucial pillars chronically inflamed; liquid pus can be expressed from both tonsils, which are submerged. Musculature of palate and pharynx normally active.

Ears. Right ear, normal findings; left ear, auricle reddened and somewhat swollen, small vesicles in various stages are seen on the helix and the central parts of the auricle, the external auditory meatus, the antitragus, incisura intertragica, and a few in front of the lobule; the external canal is reddened and inflamed but no vesicles or erosions are seen; the tympanic membrane is unaffected, landmarks clean cut and long process of anvil can be seen.

Hearing. Right ear good for conversational voice, whisper,

high and low forks. Left ear, high forks shortened; whisper 0; with Bárány noise apparatus in right ear, left ear hears conversational voice at ten feet. Weber to the right.

June 8. Facial movement has improved very much; left eye can be completely closed, altho some epiphora remains; dryness of mouth and bitter taste much lessened; eruption and swelling of auricle cleared. All of her lower teeth were extracted the week of May 17th. She refuses to have her tonsils removed.

Seven months after onset of the disease, all symptoms had cleared except for a very slight paresis of the left face, and a sensation of itching and burning at times affecting the central part of the left auricle.

In arranging cases of this nature according to their varying types, Hunt[2] has given the following classification:

I. The syndrome and its clinical types.

A. Herpes oticus.

B. Herpes oticus with facial palsy.

C. Herpes oticus with facial palsy and hypoacousis.

D. Herpes oticus with facial palsy and Meniere's complex.

II. Subgroups of the syndrome.

Herpes facialis with facial palsy and auditory symptoms.

Herpes occipitocollaris with facial palsy and acoustic symptoms.

Herpes zoster of the cephalic extremity, with auditory nerve complications.

Following this arrangement case 3 would be classed in group I under the clinical type C. Case 1 would appear under group II, clinical type 1, herpes facialis with facial palsy and auditory symptoms, but with the addition of herpes auriculae and herpes linguae.

Case 2 is one of herpes facialis, auricularis et occipitocollaris with auditory symptoms.

Of the three cases the only one to come under clean cut classification is case 3, cases 1 and 2 not coming distinctly under any one of the subgroups.

It is of interest to note that two of the three cases presented a complete paralysis of the facial nerve; this aids in confirming the fact that seventh nerve palsy is very commonly associated with herpes zoster oticus or herpes zoster of the cephalic extremity.

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DISCUSSION.

Dr. Harold I. Lillie, Rochester, Minn: My experience with herpes zoster oticus has been limited to five cases. The condition occurs in one's practice not frequently enough to be common but frequently enough to be considered in the differential diagnosis in cases that do not conform to typical clinical syndromes. In my experience, the characteristic symptoms have occurred as very severe pains over the mastoid and within the ear without physical signs to account for it, and with exquisite tenderness in the next twelve to twenty-four hours. The patients do not even permit themselves to touch the area. Shortly after the onset of tenderness, redness and well defined elevation of the skin, with or without vesicles, appear. In two cases there were vesicles.

Unfortunately, there is no particular treatment that will modify the symptoms unless ordinary sedative measures for relief of the pain are employed. Surgical interference is not indicated. One case of herpes zoster ophthalmicus has come under my observation, in which the pain was so intense for twelve hours, that the physician who referred the case had suggested that a sinus operation might have to be performed. Because there were no physical signs of frontal sinusitis, operation was deferred, and within the next twelve hours the diagnosis was perfectly obvious, as all the signs of herpes zoster appeared.

Sedatives to relieve the discomfort, and time for the disease to run its course seem to be all that we have to offer the patient.

Dr. H. B. Lemere, Omaha: The point I wish to cover in the discussion of this most comprehensive paper is the consideration of herpes in atypical cases, in which are present many of the symptoms of mastoid involvement. My experience in well developed typical cases is limited to the recognition of the one quoted below. I think at times, when complicated and obscured by other pathology, the involvement of the geniculate ganglion has been passed by unrecognized. This might be borne in mind when the pain is out of proportion to the other symptoms.

Tweedie reports a case in which there was dizziness, deafness, facial paralysis and auricular herpes, in which the drum was opaque, with a rough, glistening appearance. He thinks the herpes had disappeared. It seems more likely that the eruption was in the tympanic cavity. It it had, at any time, been external to the drum, it would seem that the remains of the spot would have been seen as dried serum and exfoliated epithelium.

In an obscure case of my own, there was a thick, glistening drum with intense pain, unrelieved by paracentesis, a sclerosed mastoid, shown by X-ray, and a slight rise in temperature. The continuance of these symptoms and the intense suffering of the patient induced me to perform a simple mastoid operation. Little was found pathologically, except bony sclerosis. The operation was followed by temporary facial paralysis. The pain gradually ceased. I am inclined, in review, to explain this case as one of herpes oticus, with the herpes confined to the tympanic cavity and not appearing in the canal, the facial, already the seat of infiltration, becoming paralyzed by the jarring of the operation.

The following case is one which was sent to the Hospital, University of Nebraska Medical College, for emergency operation on the mastoid. On entrance, the case was immediately recognized as a clear case of herpes zoster oticus.

Mrs. E. T.: Admited University Hospital, Omaha, December 15, 1919, with following history in brief: General History: No significance in regard to present complaint. Special History: One week ago, left ear began to ache. Five days later, had slight discharge, redness below and behind the ear, and complete left facial paralysis. Examination showed right ear normal. Left Ear: Drum normal, hearing undiminished. Herpetic spots on posterior surface of lobule and on the posterior inferior wall of auditory canal near drum membrane. Complete facial paralysis left side. In seeking for foci of infection: Teeth: Had all been removed, several years previous. Gums firm. Nose and Pharynx: Showed no infectious secretion. X-ray, all sinuses clear. Tonsils: Reddened and surrounding inflammation. Gaseous matter expressed. Bad odor on breath.

The pain subsided in four weeks, and the facial paralysis in about six weeks. The tonsils were removed and it seemed to us that the tonsils, which were very foul, might be the cause of the herpes.

Dr. Frank L. Dennis, Colorado Springs: The interesting part of this paper to me is the fact that cases of herpes zoster may arise without the herpes. This one point, as brought out by Dr. Sears, has been stressed several times in the literature, and to me it is most interesting.

I think there can be no doubt that many of these cases of sudden, transient paralysis, perhaps with pain and perhaps without, are cases in which the virus of infection, whatever it may be, has involved the posterior root of the ganglion.

In looking at the question of the etiology of this disease, the work of Teague and Goodpasture interested me. They published their work in August of 1922 and 1923. They were able to produce in animals, by inoculation with herpes simplex, a disease identical with the human herpes zoster. They also proved that the virus of herpes can pass along the nerves to the ganglion. Some time, I presume, we will learn what this infective virus is, but this fact, which apparently has been proven, that this virus can pass along the nerve sheath from the periphery to the center, is to me most astonishing.

A NEW PROCEDURE IN THE TREATMENT OF CHRONIC MAXILLARY SINUS SUPPURATION IN CHILDREN.*

ARTHUR M. ALDEN, A.M., M.D., F.A.C.S.

ST. LOUIS, MO.

In the treatment of chronic maxillary sinus suppuration in a child, we are mainly concerned with the problem of securing adequate drainage, and some means by which these sinuses may be frequently and comfortably irrigated. Dean conducts all sinus treatments in children under ethylene anesthesia. General narcosis, no matter of what type or what the anesthetic agent may be, is never without danger, and the thought of administering from six to twenty general anesthesias to a child is not a pleasant one.

Shea, in a paper read two years ago, suggested opening the antra underneath the inferior turbinate and inserting a rubber tube, thru which the sinus could be irrigated daily with no discomfort to the child. On the face of it, this seemed a very rational procedure, but when put into practice, certain technical difficulties immediately became manifest. In the first place, the insertion of a straight rubber tube around the corner thru a child's small nostril is not as easy as it sounds. Second, the straight tubes, even tho once comfortably placed in the sinus, slip out very easily, and it is almost impossible to replace the tube without a general narcosis. The added danger of the tube becoming dislodged from the sinus and becoming a foreign body in the lung must always be kept in mind.

With a view to avoiding the objectionable features associated with the use of the straight tube, I had made a small rubber tube with a soft rubber flange, so that its accidental displacement is almost impossible. In order to make the insertion of the tube easy, an obturator was devised with a slide, so that when the flanged end of the tube is in the antrum, the tube can be held in position while the obturator is withdrawn

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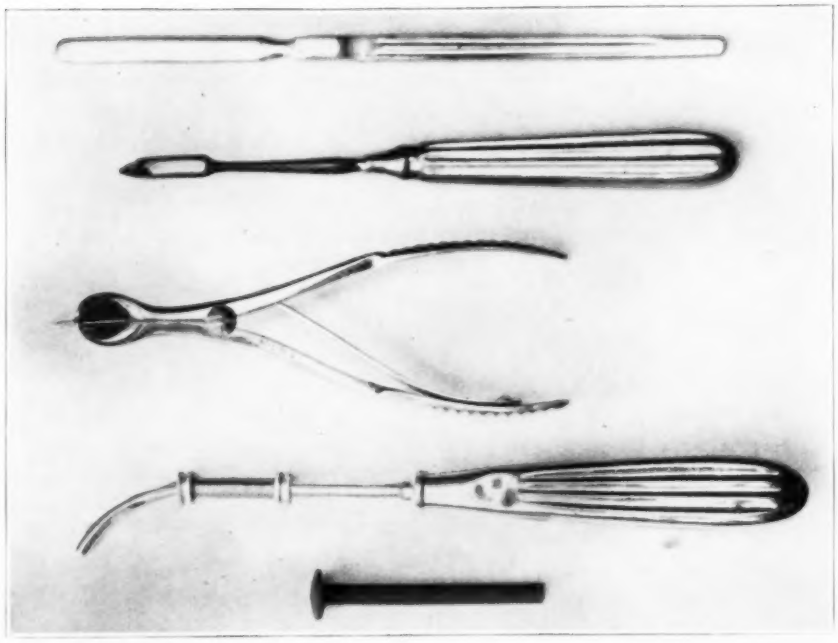


Fig. 1. Instruments used in maxilliary sinus intubation.

Fig. 2. The withdrawal of the obturator from the tube.

The instruments necessary for antrum intubation are a flat Killian elevator, a nasal speculum, a Bishop antrum perforator and the antrum tube with the obturator. Under gas anesthesia, the anterior tip of the inferior turbinate is slightly infracted and raised up by means of the flat Killian elevator. The antrum perforator is inserted, and an opening made about two-thirds of the way back on the nasoantral wall. The obturator carrying the tube is then passed along underneath the inferior turbinate, and the flanged end of the tube pushed into the antrum. The tube is held in position by means of the slide on the obturator, while the obturator itself is

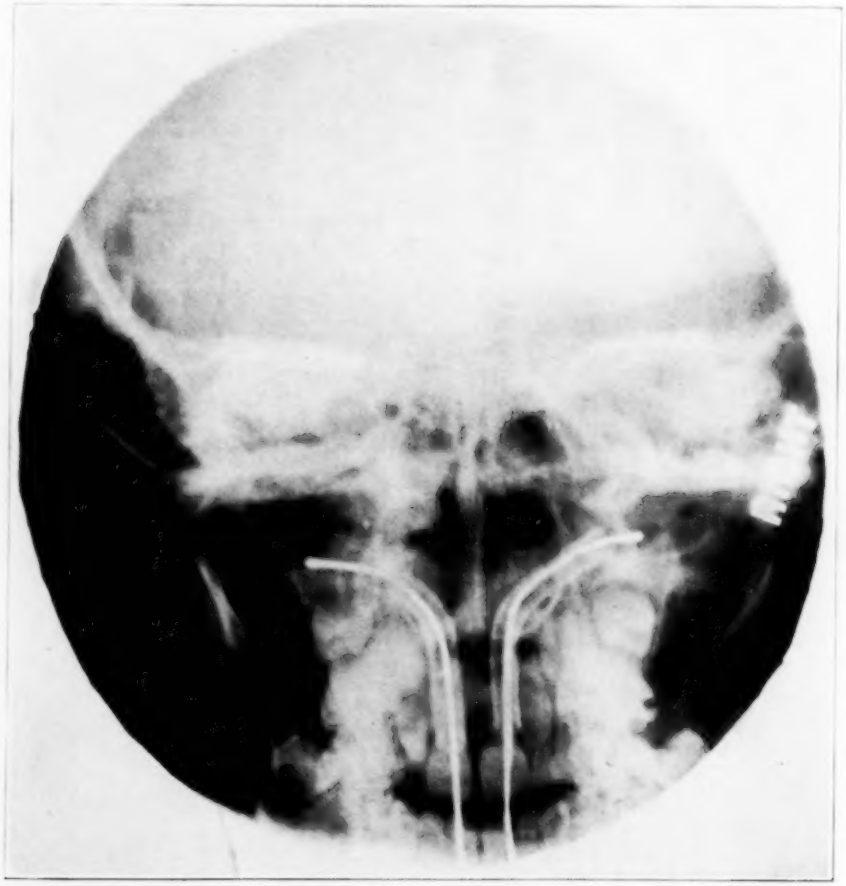


Fig. 3. Antero-posterior X-ray showing tubes in position and probes in the maxillary sinuses.

withdrawn. The tip of the turbinate is then pressed back downward, and the operation is completed.

The following day, a curved, ball pointed probe is passed thru the tube into the antrum to be sure that its lumen is not occluded with blood or secretion. Irrigation is carried out by means of a bulb syringe to which is attached a glass medicine dropper tip. After thoro irrigation, the antrum is flushed with one per cent mercurochrome or two per cent silver nitrat. This procedure is carried out daily until the sinus is free of secretion. This, as a rule, requires about ten days to two weeks.

In my series of cases, only two mishaps have occurred due to the use of these tubes. In one child, in whom for some reason or other the intubation was more than ordinarily difficult, there occurred a transient edema and ecchymosis around

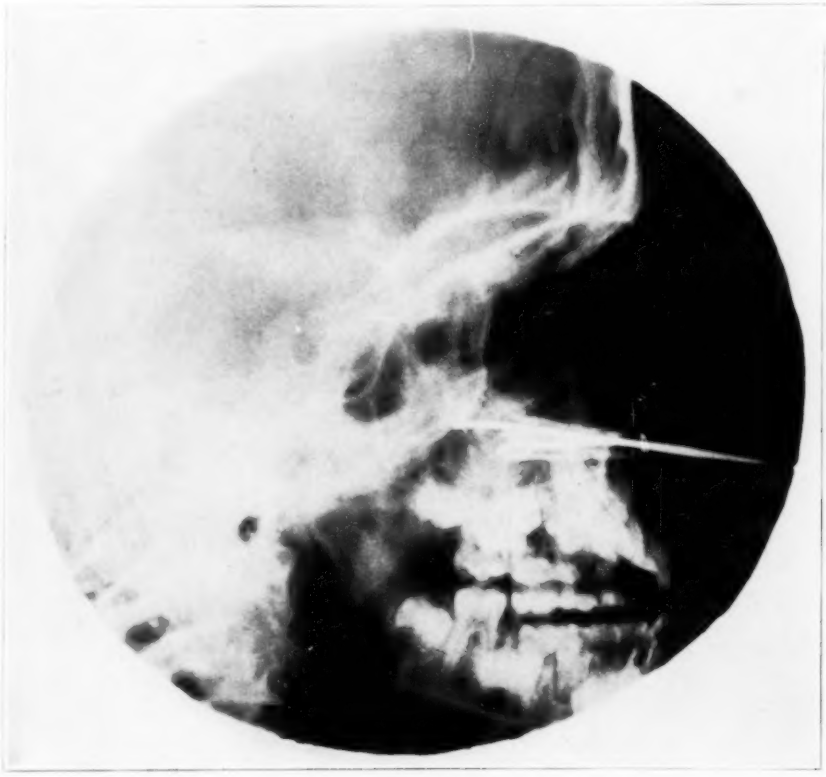


Fig. 4. Lateral X-ray showing tubes and probes in the maxillary sinuses.

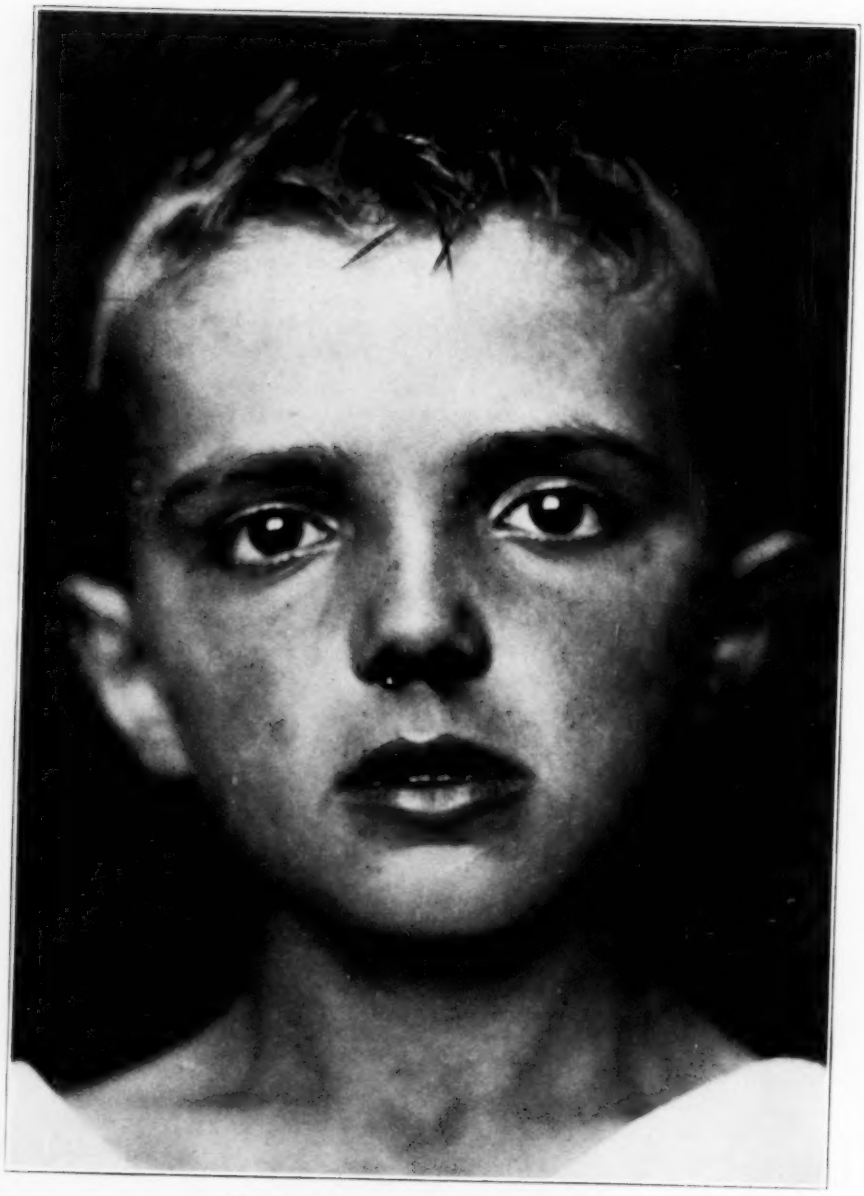


Fig. 5. Child with tubes in the sinuses.

the left eye. This disappeared in a few days. Another child developed a unilateral otitis media, which cleared up four days after incising the drum.

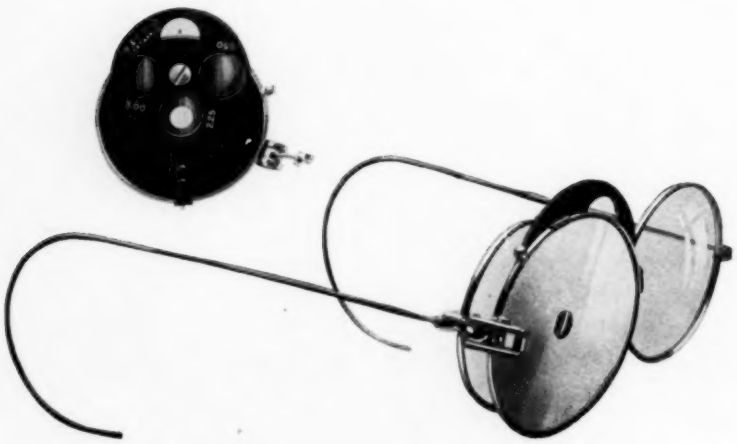
Fig. 6. The technic of irrigation.

HEAD MIRROR.

H. B. LEMERE, M.D.

OMAHA, NEB.

I wish to exhibit a spectacle head mirror, the experimental model of which, I showed last year. This is now being manufactured and is available. Its advantages over the band head mirror are: 1. Lightness, due to small size and no metal back 2. No pressure or disarrangement of hair from head band. 3. Can be used with or without lenses in frames, 4. Battery of lenses can be attached or left off, replacing accommodative effort. I have used it, as Dr. Harry Gradle suggested, for indirect fundus examination. 5. Can be swung to one side, over temple, when not in use. 6. Is easy to carry in spectacle case



PHOTOGRAPHY OF THE FUNDUS.

ROBERT VON DER HEYDT, M.D.

CHICAGO, ILLINOIS.

The fundus of the human eye has been successfully photographed for many years. To do this with ease and a certainty of success, is a very recent advancement. Because of this, ophthalmology is entering a new era, in which photographing the fundus may be put to practical daily usefulness and clinical application.

In 1922, at the International Congress of Ophthalmology in Washington, Nordenson showed photographs he had taken of the fundus. In his camera were applied the principles of simple reflexless centric ophthalmoscopy of Gullstrand. Since then the camera has been simplified until we now have available a practically fool proof apparatus. With it, one can photograph the fundus almost as easily as is the taking of a snapshot. The magnification is about 3½x linear, depending on the refraction. The time of exposure averages one-half second.

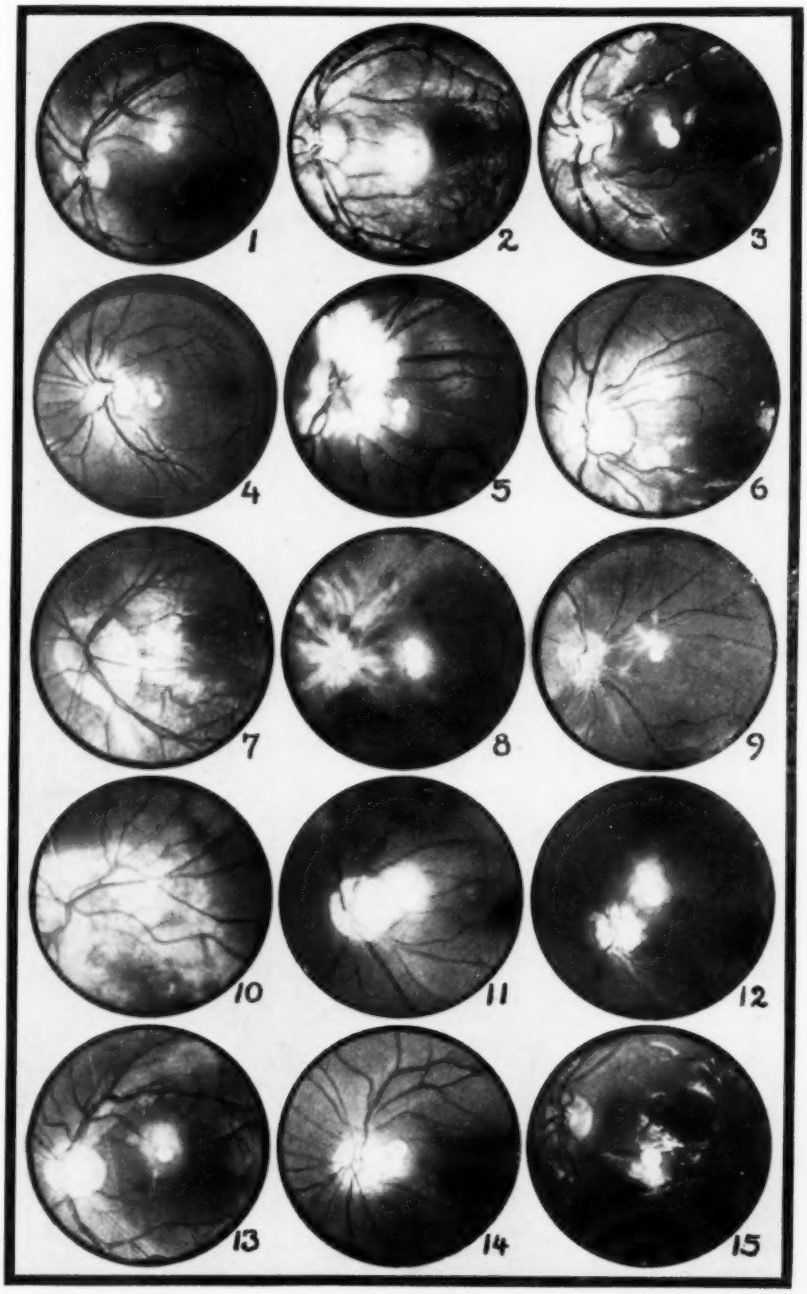
The two central spots seen in the picture are the unavoidable reflections of the microarc carbons. The detail of interest can be so placed as to avoid this central area.

Dilatation of the pupil and clear media are necessary. Very transparent anterior retinal layers, as we have them in youth, favor the distinctiveness of retinal detail. I have made exposures successfully in the presence of nystagmus.

I have the privilege of presenting these, the first fundus photographs taken in this country. The pictures of which this series is a part, were taken in my office during the past few months.

Photographs of the Fundus Taken with the Nordenson-Zeiss Camera.

1. Normal fundus.
2. Normal fundus with physiologic cupping.
3. Normal fundus showing cilioretinal vessels.
4. Normal fundus, inverted (temporal) nerve entry.
5. Normal fundus with medullated nerve fibers.
6. Myopic fundus.
7. Myopia 13 D; staphyloma, preretinal hemorrhage over the macular area.
8. Thrombosis with multiple hemorrhages.
9. Neuroretinitis; connective tissue formation; an obliterated vessel near disc. (Case of tubercular kidney.)
10. Choroiditis—traumatic from shell injury.
11. Optic atrophy—tabetic—central vision 20/30.
12. Optic atrophy following embolism. Note the collateral engorgement of several cilioretinal vessels within the disc border.
13. Optic atrophy after retrobulbar neuritis.
14. Optic atrophy in Leber's disease (hereditary optic atrophy).
15. Hole in the macula in a young girl, 7 years after perforating trauma.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FOLDING BEEBE LOUPE.

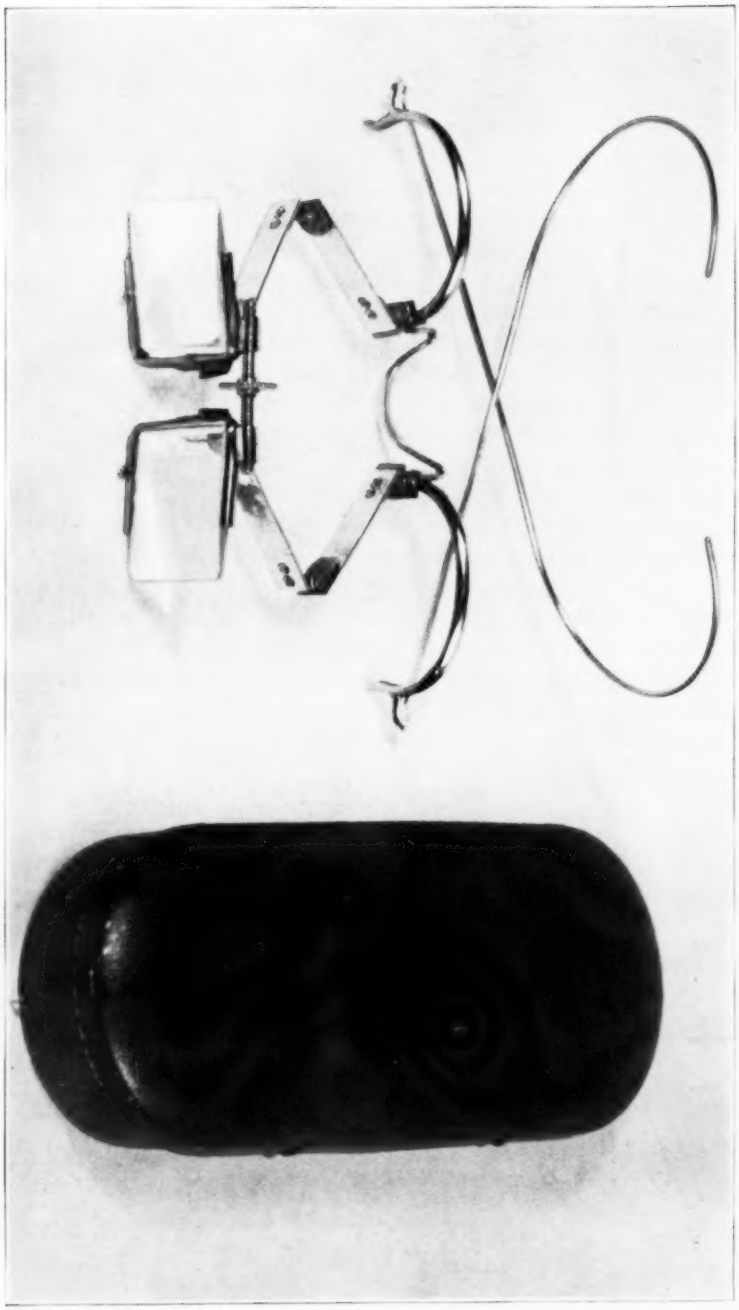
GORDON F. HARKNESS, M.D.

DAVENPORT, IA.

I have found the Beebe loope a desirable adjunct in my work. I often wished that I had one with me when away from the office and hospital.

The size of the case containing the ordinary loupe is a disadvantage when thrown in one's bag, for which reason I have devised this folding Beebe loupe, which I have found quite convenient to carry in an oculist's bag.

This was made in the local shop of the American Optical Company for me, and as yet to my knowledge is not on the market.



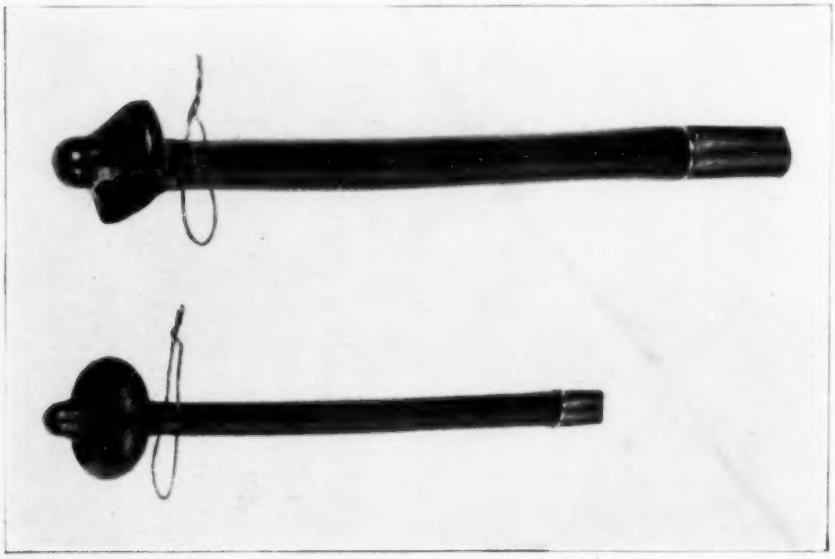
IRRIGATION AND DRAINAGE TUBE.

GORDON F. HARKNESS, M.D.

DAVENPORT, IA.

I present this tube because it has served me best in affording drainage from the maxillary antrum, and as an irrigating tube to wash out infected material in the cavity.

It consists of the Pezzar self retaining catheter, size according to the individual case, with the bulbous end cut away on two sides. Sterling silver wire, size 43/100 mm., pierces the wall of the tube on opposite sides as per illustration. The two ends are then twisted together, and the wire loop serves as a retaining shoulder to prevent the tube from slipping into the antrum beyond the point desired. The tube is threaded on a heavy curved applicator, and is then pushed into the antrum thru the inframeatal opening previously made. The applicator is then withdrawn from the catheter, the bulbous end of the cathater preventing it from slipping out. The silver wire can be bent and laid against the outer inframeatal wall, and causes practically no discomfort. The catheter, after being placed, is cut off so that the small end lies along the floor of the nasal cavity and just protrudes from under the inferior turbinate. I have felt, that by such a contrivance, one could maintain prolonged antrum drainage and irrigation, when desired, with the least discomfort to the patient.



AN OPHTHALMIC IRRIGATING BASIN.

HARRY S. GRADLE, M.D.

CHICAGO, ILLINOIS.

The basin depicted herewith is for the purpose of carrying off the drain fluids after an irrigation of the eye. The inner aspect of the basin is cut away, and is replaced by ordinary dental rubber dam which is held firmly in position by a spring clip. By pressing this aspect of the basin against the cheek of the patient, the rubber dam adapts itself to the facial contour perfectly, no matter in what position the head is held. The fluid, caught in the basin, is carried off by a rubber tube either to a sink or to a bottle of sufficient size that may be held by the patient. The great advantage presented is that none of the irrigating fluid trickles down the patient's cheek to unwanted parts.

BRONCHOSCOPE SHIELD.

HOWARD L. STITT, M.D.

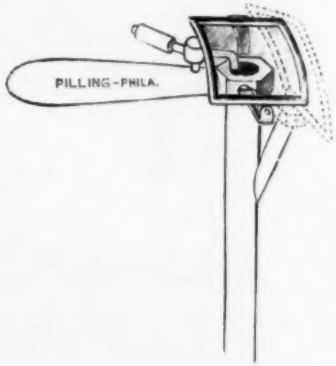
CINCINNATI, OHIO.

In the course of most bronchoscopic examinations, there is usually considerable coughing accompanied by much expectoration, which hinders quick and skillful manipulation of the instrument, aside from the unpleasantness and even danger to which the physician is exposed. To overcome these difficulties a shield has been designed.

It consists of a rectangular, metal frame with grooved edges and a convex surface, which may be attached to the ordinary Jackson scope or any scope having a similar entrance, with two flexible and adjustable arms terminating in small pins which fit into small holes drilled in the scope. It is necessary only to slip the ends of the flexible arms into the scope. Thus, a universal instrument is made.

The shield can then be raised or lowered respectively for the introduction of an instrument or for observation, as per illustration. The removable window is made from a piece of undeveloped, washed, Eastman X-ray film, fitted into the grooves of the frame. When secretions collect on this window, it may be readily cleaned with moist gauze, or another quickly inserted.

The use of this shield in connection with glasses generally worn by the physician, gives him double protection, aside from the valuable aid to his speedy and skillful manipulation.



A NEW SET OF TEST LETTERS FOR NEAR AND FOR DISTANCE VISION.

ALBERT C. SNELL, M.D.

ROCHESTER, NEW YORK.

These letters are the usual block letters constructed on the principle of the one minute limbs and five minute characters. The specific letters chosen are those recommended by the Jackson report of the A. M. A. The new feature of this new set of test letters consists in the fact, that they are so constructed, that each gradation from 20/20 Sn. to 20/200 Sn. represents exactly a 5% loss of central vision. The percentage values used are those determined by the principles laid down in the report of the Committee of the A. M. A. on Compensation for Ocular Injury. Therefore, these test letters have an equal geometric ratio for each gradation.

Most of the test letter cards now used do not contain test letters between 20/100 and 20/200. This is a comparative jump of 50%. The set of test letters presented contains five additional gradations between 20/100 Sn. and 20/200 Sn. This permits of the accurate determination of loss of central vision between these points.

The test card for determining near vision is constructed on the same principle as the distance test letters—each gradation representing a difference of 5%. These cards have been constructed by the Bausch & Lomb Optical Co. and will be distributed by them. This company have also printed industrial visual fields and industrial motor field charts, as suggested by the Committee on Compensation of the A. M. A. These charts are recommended as record charts for loss of field vision and for loss to motor fields.

TRANSACTIONS

OF THE

THIRTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

NECROLOGY
MINUTES
DIRECTORY
INDEX

DEATHS

Dr. Alexander Duane, New York City

Dr. Marcus Feingold, New Orleans, La.

Dr. Lee M. Francis, Buffalo, N. Y.

Dr. James M. Guthrie, Meridian, Miss.

Dr. Edward P. Hall, Kansas City, Mo.

Dr. George F. Keiper, Lafayette, Ind.

Dr. Nils E. Remmen, Chicago, Ill.

Dr. Otis D. Stickney, Atlantic City, N. J.

Dr. George O. Suffa, Boston, Mass.

Dr. Oliver Tydings, Chicago, Ill.

Dr. S. Lewis Ziegler, Philadelphia, Pa.

MINUTES.

September 13, 1926.

The Thirty-first Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology was held at the Antlers Hotel, Colorado Springs, Colorado, September 13 to 18, 1926.

The Monday evening meeting, September 13, was a special scientific session, at which PROFESSOR ROBERT H. GAULT of Washington, D. C., demonstrated by lantern slides and charts, "Hearing By Touch."

For paper and discussion see pages 25-34.

DR. A. A. HAYDEN of Chicago presided at this session.

September 14, 1926.

The Tuesday morning session was called to order at 9:00 o'clock by the President, Dr. Edward C. Ellett, Memphis, Tennessee.

Dr. Frank L. Dennis, Colorado Springs, Chairman of the Local Committee, made announcements in regard to various entertainments, following which the President introduced the guest of honor of the Academy, Mr. Basil Graves of London.

The President announced that the International Typographical Union was meeting in Colorado Springs at this time, and asked the Secretary to convey the greetings of the Academy to this organization.

The business session of The American Academy of Ophthalmology and Oto-Laryngology was held Tuesday evening, September 14.

The session was called to order at 8:15 by the President, DR. E. C. ELLETT.

Moved by DR. HORACE E. NEWHART that the minutes as published in the Transactions be approved as printed. Motion seconded and carried.

Report of Secretary.

Mr. President and Fellows: The membership roll of the Academy at this time includes 1,543 names. We have lost 11 members by death and one by resignation.

An effort has been made during the past two years to obtain complete records of the date of admission of each member. For this purpose, blanks were sent out with the Transactions for the last two years, and Fellows have been asked to supply this information and forward same to the office of the Secretary. As many have failed to return these blanks, the matter is again brought to the attention of all who have failed to supply the information. Prompt response will aid the officers in bringing our records and statistics up to date.

The perennial inquiry as to why the Transactions are not forthcoming, is with us again this year. The same answer applies as in former years, in most instances. The Transactions are sent to all members free of charge if dues for the current year are paid on or before March 1st of that year.

It is a source of great satisfaction to your officers to be able to announce, that during the year, part of the income from the Research Fund, which has been steadily growing, was devoted to a Fellowship in Ophthalmology. Dr. D. B. Kirby of New York conducted a research study on "The Origin and Nutrition of the Crystalline Lens," the result of which he presented in a formal communication which appears in our program. In the back pages of the program, a form of bequest to the Academy will be found. It is true that we now have a modest sum available for research work, but it is only a beginning. The attention of the members is called to this means of helping along. It is the hope of many of the members of the Academy, that this Research Fund may in time reach such figures that the income from the same may be devoted to research work on a proper scale. A similar sum of money was voted to a Fellowship in Oto-Laryngology. Dr. William V. Mullin, now of Cleveland, has undertaken a study of great interest in otolaryngology. This year, therefore, there has been the beginning of what promises to be the most advanced and valuable phase of the Academy's work, namely, an active research department.

The increasing activities of the Academy demand slight changes in the Constitution and By-Laws. These changes,

which will be presented for your consideration, are largely technical and changes in terminology, but essential to the reorganization policy, and therefore deemed advisable by the officers and members of the Council.

Finally, a word to those whose letters failed to reach my office, and to those who failed to receive letters addressed to them from the Secretary's office.

During the past year and part of 1925, many letters failed to reach me, and many letters sent from my office failed to reach their destination. Much unjust criticism has been directed against the Secretary because it is alleged that he has not answered his correspondence. The trouble is not in the Secretary's office, but in the United States mails. The matter has been taken up by the United States Mail Department in Washington and in Philadelphia. Up to the present time, the source of the trouble has not been uncovered, but an honest effort is being made to locate and correct the trouble. As this question applies generally to my own private correspondence, as well as to that of the Academy, I feel that it is but fair to me to include this statement in the Report of the Secretary.

Respectfully submitted,

LUTHER C. PETER.

Moved by DR. GEORGE F. SUKER that this report be accepted and placed on file. Motion seconded and carried.

CONDENSED REPORT OF TREASURER.

Cash Balance, Oct. 6, 1925	$ 5,296.77
Receipts	30,469.99
TOTAL	$35,766.76
Disbursements	$27,080.82
CASH BALANCE	$ 8,685.94
Bonds	$64,032.60
TOTAL	$72,718.44

Respectfully submitted,

SECORD H. LARGE.

REPORT OF PUBLIC ACCOUNTANT.

Union Trust Building
Cleveland.

August 28, 1926.

The American Academy of Ophthalmology and Oto-Laryngology, Cleveland, Ohio.

Dear Sirs:

In accordance with instructions received, we have examined the accounts of your Academy for the period from October 6, 1925 to July 31, 1926, and submit herewith a summary of receipts and disbursements for the period, together with a statement of the assets at July 31, 1926. We also attach a schedule of bonds owned at the latter date.

All recorded receipts were traced to the credit of the Academy's bank account and properly approved vouchers were produced for all disbursements. The balance in the bank at July 31, 1926 was verified by certificate from the bankers and the bonds on hand at that date were counted by us.

Yours very truly,

Price, Waterhouse & Co.

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SUMMARY OF RECEIPTS AND DISBURSEMENTS FROM OCTOBER 6, 1925 TO JULY 31, 1926.

Cash in bank October 6, 1925.....		$ 5,296.77
Receipts:		
On Income accounts:		
Dues from members and applications for membership.....	$14,670.00	
Income from exhibitors at annual meeting	2,360.48	
Interest on bonds, exchange, etc.....	3,204.51	
Profit on bonds sold.....	481.50	
	$20,716.49	
On Capital Accounts:		
Bonds sold—cost.....	9,753.50	
Total receipts.....		30,469.99
		$35,766.76

Disbursements:		
On Expense Account:		
Office expense	$ 2,899.56	
Expense of annual meeting	7,847.65	
Exhibition expense	785.11	
Typewriter purchased	102.50	
Sundries	69.25	
Interest accrued on bonds purchased	169.75	
	$11,873.82	
On Capital Account:		
Bonds purchased	15,207.00	27,080.82
Cash in Bank July 31, 1926		$ 8,685.94

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SCHEDULE OF BONDS OWNED JULY 31, 1926.

Description		Par Value	Cost
Kansas City Fort Scott & Memphis Railway — Refunding General	4 %—1936	$ 5,000.00	$ 4,352.50
Chicago, Rock Island, and Pacific Railway— Refunding General	4 %—1934	6,000.00	5,337.00
American Telephone & Telegraph Company Debentures	5½%—1943	4,000.00	4,026.00
Southern California Edison Company	5½%—	4,000.00	3,865.00
Columbia Gas & Electric Company 1st	5 %—1927	4,000.00	3,951.00
Denver Gas & Electric Company	5 %—	4,000.00	3,951.00
Youngstown Sheet & Tube Company	6 %—1943	5,000.00	5,135.00
Midvale Steel & Ordnance Company, Convertible—Sinking Fund	5 %—1936	5,000.00	4,735.00
Swift & Company — Sinking Fund Gold Notes	5 %—1932	5,000.00	4,845.00

Witherbee Shuman Company	6 %	5,000.00	4,925.00
Van Swearingen Company	7 %	9,000.00	8,910.00
Union Square Company, Cleveland	6½%	5,000.00	5,000.00
Seventeen Twenty Euclid Building, Cleveland	7 %	5,000.00	5,000.00
		$66,000.00	$64,032.50

SUMMARY OF ASSETS JULY 31, 1926.

	Oct. 6,1925	July 31, 1926	Increase
Cash in Bank	$ 5,296.77	$ 8,685.94	$ 3,389.17
Bonds owned, per schedule	58,579.00	64.032.50	5,453.50
	$63,875.77	$72,718.44	$ 8,842.67

The foregoing increase in assets reflects the excess of income receipts over expenses as follows:

Income receipts as above	$20.716.49
Expenses for period as above	11,873.82
	$ 8,842.67

REPORT OF SENIOR MEMBER OF COUNCIL.

I first wish to report on the amendments to the Constitution and By-Laws as published in the program. The Council makes the following recommendations:

1. That thruout the Constitution and By-Laws, wherever the word "Secretary" occurs, the word "General" precede it.

The Council recommends this change.

2. Constitution, Article 4, under "Officers:" After the words "Editor of the Transactions," insert the following: "Secretary of the Section on Ophthalmology, Secretary of the Section on Oto-Laryngology, Secretary of the Section on Ininstruction."

The Council recommends that this be included in the changes in the Constitution.

3. Constitution, Article 4, under "Officers:" After the words "at a regular meeting" insert the following: "The General Secretary and the three Section Secretaries shall be

elected at an annual meeting, each to serve for a period of three years. The General Secretary shall be eligible to reelection for a second period of three years."

The Council recommends that this be amended as follows:

Constitution, Article 4, under "Officers:" After the words "at a regular meeting" insert the following: "The General Secretary shall be elected annually; the three Section Secretaries shall be appointed annually by the President with the approval of the Council."

4. By-Laws, Article 4, under "The General Secretary:" After the words "He shall assist the President" insert the following: "and the Section Secretaries."

The Council recommends this as published.

5. By-Laws, Article 4, under "The General Secretary:" After the words "publication and distribution" insert the following paragraph: "He shall send out all statements and shall collect the annual dues of the Academy, and shall forward to the Treasurer all money so collected."

The Council recommends this as published with the addition, "He shall give bond of $2,000, the expenses of same to be borne by the Academy."

6. By-Laws, Article 4, under "The Treasurer:" Omit the words "He shall keep a correct list of the members who have paid their dues, and notify the Secretary of all delinquents."

This is recommended as published.

7. By-Laws, Article 4, after "The Editor of the Transactions:" Insert the following paragraph, headed "Section Secretaries:" "The three Section Secretaries, with the assistance of the President and the General Secretary, shall arrange the scientific program of the Academy. They shall also assist the presiding officer of each scientific session."

The Council recommends that this be omitted entirely.

8. By-Laws, Article 4, under "The Program Committee:" Correct paragraph to read as follows: "The Program Committee shall consist of the President, General Secretary, and the three Section Secretaries. It shall prepare the scientific program of the annual meetings."

The Council recommends a slight change in this, namely, to insert after the word "President" the words "First Vice-President."

9. By-Laws, Article 4, under "The Committee on Arrangements:" After the words "it may deem expedient" insert the

following: "This Committee shall consist of the General Secretary, the Treasurer, and the Chairman of the Local Committee."

This is recommended as published.

10. By-Laws, Article 4: Omit paragraph headed "The Committees."

The Council recommends the substitution of the following: "The Committee on Pathology shall consist of five members to be appointed annually by the President."

11. By-Laws, Article 5, under "Dues:" In last paragraph substitute for the words "Treasurer or Secretary" the words "General Secretary."

This is recommended as published.

12. By-Laws: Insert the following paragraph, headed "Business Session," as Article 7: "The Regular Business Session shall be held on the evening of the first day of the annual meeting of the Academy. If deemed advisable by the Council other business sessions may be called at the beginning of a scientific session."

This is recommended as published.

13. By-Laws: Change "Article 7" to "Article 8."

This is recommended as published.

14. By-Laws: Change "Article 8" to "Article 9."

These are to be omitted as unnecessary.

15. By-Laws: Article 9, under "Order of Business."

a. In item "7" omit the word "Standing."

b. In item "7" omit the word "Arrangements."

c. Omit items "8" and "9."

d. Make new item "8" as follows: "Report of Committee on Arrangements."

e. Make item "10" item "9."

f. Make item "11" item "10."

g. Make item "12" item "11."

h. Insert the following as item "12:" "Election of Members."

This is recommended as published except that the Report of the Senior Member of Council should appear after the Report of the Editor of the Transactions.

OFFICERS FOR THE ENSUING YEAR.

President-elect	Dr. Luther C. Peter	Philadelphia, Pa.
1st Vice-President	Dr. H. W. Woodruff	Joliet, Ill.
2nd Vice-President	Dr. Ralph A. Fenton	Portland, Ore.

3rd Vice-President	Dr. Conrad Berens	New York City, N. Y.
Treasurer	Dr. Secord H. Large	Cleveland, O.
General Secretary	Dr. W. P. Wherry	Omaha, Neb.
Editor of Transactions	Dr. C. Loeb	Chicago, Ill.
Member of Council (3 years)	Dr. Frederick L. Stauffer	Salt Lake City, Utah
American Board for Ophthalmic Examinations	Dr. F. P. Calhoun	Atlanta, Ga.

As to the place of meeting, we have invitations from Detroit and from Montreal. We place them before you that you may make your choice. Respectfully submitted,

THOMAS E. CARMODY.

REPORT OF COMMITTEES.

The Program Committee offered the program as its report.

REPORT OF CHAIRMAN OF SECTION ON INSTRUCTION.

The Section on Instruction has no very definite report to offer, inasmuch as our report is comprised in the program that has been sent to you, and that is to be given on the last two days of the meeting.

The Committee on the Section on Instruction has felt that perhaps a slight change might be desired in the character of the instruction work for the future—that the members are rather fed up on didactic instruction—and it has in mind the possibility of endeavoring to offer some group instruction of a semiclinical nature in the future, something on the order of the Round Table, which went so well last year, and we hope will be equally well received this year. The details are still in the rough, but we believe there is something to be gained by groups meeting informally with their instructor and discussing the matters among themselves, rather than sitting down in a large hall to listen to a didactic lecture. Apart from that, the committee merely wishes to express to the Academy and to the officers its thanks for all the support it has received.

HARRY S. GRADLE.

REPORT OF COMMITTEE ON PUBLICITY, SERVICE AND LEGISLATION.

There was only one thing before the committee, but unfortunately it was lost in the mails, and therefore no action

was taken. There was to have been a meeting in which Dr. Francis was to represent us. Whether that meeting took place, we cannot say, because the unfortunate circumstance of Dr. Francis' death robs us of the possibility of his telling us what might have taken place. We therefore have no report to make.

DR. THOMAS E. CARMODY.

REPORT OF GENERAL COMMITTEE ON OPHTHALMIC AND OTO-LARYNGIC PATHOLOGY.

The Section on Ophthalmic and Oto-Laryngic Pathology has been fairly active during the past year, as evidenced by the comparatively large number of specimens that have been sent in. Altho we recapitulate each year what this section is, still it seems in many instances it has failed to penetrate, and I would like to bore you briefly if I may.

Under an arrangement between the Army Medical Museum at Washington, the American Academy of Ophthalmology and Oto-Laryngology, the American Ophthalmological Society, and the Section on Ophthalmology of the American Medical Association, the Army Medical Museum is carrying on work in ophthalmic and otolaryngic pathology, and will gladly accept any specimens sent in, and if accompanied by the history, will work them up histologically and have them diagnosed by the most competent pathologists of the country. Diagnosis and observation will be returned to the donor of the section, as well as one or more sections of the specimen. The Army Medical Museum by doing this obtains specimens that would be entirely without their possibility. The donors, on the other hand, can obtain this beautifully done pathologic work, and have a diagnosis made in a way that it would be very difficult to obtain in an outside manner. Unfortunately, not enough members are taking advantage of this, because it is hoped that in the future the Army Medical Museum will be able to offer a museum of ophthalmic and otolaryngic pathology second to none in the world. Eventually, the Museum will prepare study sets of 100 or 200 characteristic specimens, that will be loaned to men thruout the country for a period of three or four months as they desire, in order that men may carry on their pathologic studies at home without having to leave their work.

As to the character of the material received, we have been obtaining gross specimens. These specimens have been diagnosed, and have been aded to the Museum collection. Other items, such as models and so forth, have been added to the collection—such as the Ball collection. In the six years that the Museum has been cooperating with us, we have received 570 eyes that have been sectioned and diagnosed, and returned to the donors. Other items have been received, and altogether, in the six years, we have received museum specimens to the number of 1,883. It is possible that in the near future financial assistance may be required to maintain this, and eventually the Academy may be called on for a year or two to supply a deficiency that should be supplied by the Surgeon-General's office, but which unfortunately cannot be supplied owing to our parsimonious Congress.

HARRY S. GRADLE.

This report was approved as presented.

REPORT OF AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS.

The Board has held at Denver, this year, its twentieth examination, and we have certificated up to date something like 525 persons in the United States. The examinations have been conducted this year as in years past, and we feel that the influence of the Board is more widely felt now than it has been. The character of the condidates who come before the Board is considerably higher than it has been in the past, and we think the standards achieved have been higher. We think we will still further widen the influence of the Board by following the example set by the Oto-Laryngic Board, namely, the issuing of a directory of all of the certified persons. That directory will soon be out. It will be arranged geographically and alphabetically, and will be very convenient for reference by any persons who desires to know who may be certified by the Board and the locality in which he lives. We feel in this way it will be a great help to those who have already been certified, and will probably be a stimulus to others who have not been certificated, to make application for a certificate, because such directories as that of the Oto-Laryngic Board will be in possession of the various physicians, and will also be in hospitals and medical schools.

We held a special examination in San Francisco after the meeting of the American Medical Association at Dallas, and

probably next year we shall hold a special examination at Spokane, Washington, since I understand the Oto-Laryngic Board will hold such an examination there.

W. H. WILDER, SECRETARY.

The above report was approved as presented.

REPORT OF AMERICAN BOARD OF OTO-LARYNGOLOGY.

We held an examination at Denver, and had 35 candidates. As you know, this Board has representatives from the American Laryngological Society, the American Otological Society, the American Triological Society, as well as the Section on Oto-Laryngology of the American Medical Association, so Dr. Wherry and myself simply represent the Academy on that Board.

During the last year we have published a directory, as Dr. Wilder has told you, of those already certificated. That of course does not include those examined at Denver, at San Francisco, or at the Montreal meeting of the Laryngological, Otological and Triological Societies. Nor does it include those examined yesterday, but these will be in the new book, which will probably be published next year. I am glad to hear that the Ophthalmologic Board will publish such a book on their own account. We have had the Ophthalmologic Board as an example before us, and they have helped us a great deal, so we are glad that we have put one over on them by publishing a directory before they did.

THOMAS E. CARMODY.

This report was approved as read.

COMMITTEE ON EYE LEGISLATION.

No report. Committee continued.

REPORT OF SPECIAL COMMITTEE ON TUNING FORKS AND TESTS.

To the Members of the Section on Oto-Laryngology of the American Academy of Ophthalmology and Oto-Laryngology:

As stated on previous occasions, this Committee was appointed at the meeting of the Academy in Philadelphia, in October, 1921, and has made a report every year since then with reference to the standardization of tuning forks and tun-

ing fork tests. In 1925, a rather detailed report was given at the meeting in Chicago, and since this as well as the other reports are on file and may be consulted by those who are interested, it seems that a more condensed statement will suffice this year. There are several points, however, that the Committee would like to emphasize, viz:

One of these is the fact that your Committee has had the privilege of cooperating with Major Reasoner of the United States Army in the effort to draw up proper specifications for sets of tuning forks to be used in the various army units. The material and construction of forks was considered, as well as the selection of a minimum set to be used. The latter included five forks, the lowest C, 64 double vibrations, c-1, 256 double vibrations, c-4, 2048 double vibrations, and a pair of a-1, 435 double vibrations forks. The first three forks are to be used in the diagnosis of disturbances of hearing, and the latter two in the Stenger test for the determination of malingering. The Committee feels gratified to think that in a modest way at least it has been of some service to our government.

It was mentioned in our report of 1925, that the Bureau of Standards at Washington is prepared to determine the so-called "damping constant" or decrement of the fork. For a fee of $2.00 each, the forks which are sent to the Bureau will be standardized and a certificate furnished giving the "damping constant" of each fork. The advantage of knowing this "damping constant" is that it is then possible to determine the percentage of loss of hearing as figured in sensation units. The average length of time which the fork is heard by a normal individual is used as the standard. The length of time which the fork is heard by the individual under examination is subtracted from this amount, and the remainder multiplied by the socalled "constant" of damping, thus giving the percentage of loss of hearing. At least one of the members of the Committee has had some of his forks thus standardized, and now uses them to get a definite figure with reference to the loss of hearing, just as one would with an audiometer. However, to get a complete curve, as is done with the audiometer, would require longer time. On the other hand, with forks, one can test the bone conduction, which at the present time cannot be successfully carried out with the types of audiometers now on the market.

The forks are to be held a uniform distance from the ear, preferably with the wide or outer surface of the prongs facing the ear, without touching the auricle or vibrissae, in order to get the maximum intensity of sound waves, to avoid interference waves which occur at certain points, especially at the angle between the broad and narrow surfaces, as well as to prevent damping of the fork by contact with the tissues. Holding the distal end of the prongs near the ear gives the loudest sound per unit of area, because of the amplitude at that point.

So far as possible, in exciting the fork, it should be struck at the "percussion point," which lies at about the junction of the distal and middle third of the prongs, because at this place one gets the greatest intensity of sound and the least overtones. The general rule is that the lower the pitch of the fork, the softer should be the material used for striking it.

When testing bone conduction, one may either use a subjective control, by means of the examiner's own conduction, or the objective method, in which the criterion used is the average duration of time that the fork is heard by bone in a series of normals previously tested with that fork.

The Committee recommends as in previous reports that at first only three standard tests be considered, namely: The Weber, Schwabach and Rinne. The Weber and Schwabach tests are performed by placing the fork on the median line of the vertex or forehead, and for this purpose a fork fairly heavy, so that it rests largely of its own weight, is desirable. In the Weber test, one determines whether the sound is perceived in the vertex or, as the patient says, "heard in the head," or whether the tone of the fork is lateralized, that is, seems louder or entirely in one ear or the other. It has been suggested that one gets a fairly good idea of the reaction of the Weber test when the examiner designates the findings as "forks definitely lateralized to the right or left," "indistinctly lateralized to the right or left," and "not lateralized," etc.

The Schwabach test is usually performed by placing the fork in the median line of the vertex or forehead, avoiding all pressure except that of the weight of the fork. One notes the time that the sound is heard, and by occasionally lifting the fork from the head and then replacing it, the "fatigue symptom" is avoided. It is then possible to state whether the bone

conduction is "appreciably shortened," "moderately shortened," "very much shortened" or "scarcely heard," and these expressions may perhaps give more definite information to the reader than the mere designation of the number of seconds that the hearing of the fork is shortened or lengthened. If the examiner states that the fork is heard by the normal individual an average of a certain number of seconds, and that by the patient it is heard so many seconds less or so many seconds more, it will give a fairly graphic idea of the state of the bone conduction.

For the Rinne test, a medium pitched fork is usually employed, and the difference between the bone and air conduction determined. Besides giving the figures representing this relation of air to bone conduction, the use of expressions somewhat like the following will perhaps convey a clear idea to the reader regarding the state of the patient's hearing for bone and air conduction: such as "Rinne strongly negative," "strongly positive," "plus minus," "very much shortened," "positive," "moderately shortened," slightly negative," "strongly negative," etc., etc.

The upper tone limit can be further tested by means of the Galton (Edelmann-Galton) whistle, or the monochord. The latter is the only instrument with which high tones can be properly tested by bone conduction.

Your Committee, with the exception of Dr. Mackenzie, recommends that five forks should be procured by those doing functional testing, namely: C-1, 32 double vibrations, C, 64 double vibrations, c-1, 256 double vibrations, c-2, 512 double vibrations, and c-5, 4096 double vibrations per second. Mackenzie prefers for low tones a fork of 64 double vibrations; for middle tones, a fork of about 190 double vibrations, using this for the Weber, Rinne, and Schwabach tests; for the high tones, a fork of 2048 double vibrations.

The Committee wishes to emphasize the character of the forks to be employed. They should be well balanced, of rustless metal, if such can be obtained, of pitch within a reasonable range, of fairly long duration of vibrations, moderate in price and as free from overtones as possible. With some of these forks, we advise the employment of weights to eliminate the overtones.

Thru the courtesy of Mr. Newell D. Parker, one of the members of your Committee has had the privilege of seeing

forks made of aluminum. They seem to have good tones and fairly long duration of vibrations. How expensive these will be as compared with steel forks, we do not yet know. It seems to us most desirable that high grade forks of rustless metal be produced, and we believe efforts in the right direction are now being made. Perhaps the new alloy called "Sterlite" will serve the purpose. In a nickeled fork, it often happens that the nickeling later peels and adventitious sounds are caused by the loose pieces of metal. If forks are unnickeled, and are not properly oiled or otherwise protected from the air or the perspiration of the examiner's hands, oxidation or corrosion occurs, and in time will change the pitch of the fork.

Former reports have called attention of the members to the fact, that for the recording of tuning fork and other functional tests, various formulae and diagrams may be used. The acoumetric formula adopted by the International Congress in Buda Pesth in 1909, and also the system used by the Vienna Clinic are very useful. All these methods are fairly accurate if carefully and systematically used, but none of them is perfect.

During the past year or two, a number of otologists have been using audiometers, especially the type devised by the Western Electric Co. Audiometers are very expensive and with them, as they are at present constructed, hearing by air conduction, only, can be tested. The lowest and the highest limits can be determined only with the very expensive apparatus that is used more for research work. With good tuning forks, one can make an accurate diagnosis, if the forks are applied as previously mentioned in this report; and if the "damping constant" is known, one can get the percentage of loss of hearing the same as with an audiometer. Furthermore, this instrumentarium is very inexpensive, freely portable and easily manipulated. In a word, we feel that the audiometer is a very desirable apparatus, but for the average otologist, the use of a few good forks will suffice for all practical purposes.

As stated in the beginning of this report, the details of many of the recommendations have been previously presented, especially in the report of 1925, and are therefore not repeated here.

We recommend the adoption of this report and the continuance of the Committee for further study of the question.

Respectfully submitted,

L. W. Dean
E. G. Gill
George W. Mackenzie
Robert Sonnenschein, Chairman.

Report of The Committee on the Hard of Hearing.

Your committee has to report progress during the past year chiefly along the line of publicity and activities for the prevention of deafness.

Papers detailing modern methods of detecting hearing loss, and pointing out the results to be achieved thru the periodic examination of the acuity of hearing of school children by audiometric methods, have been presented and discussed by members of this committee before the American Medical Association, the American Otological Society, the American Laryngological, Rhinological and Otological Society, and the American Federation of Organizations for the Hard of Hearing. These papers have been made possible by the very recent perfection of instruments developed by the research laboratories of the Bell Telephone Company, and the Western Electric Company, whose personnel have given most generous assistance in the work. Papers dealing with this subject were also presented and demonstrations made before smaller groups of medical men and educators.

Thru cooperation with the members of like committees of the three other national organizations interested in Otology, the following resolution has been adopted by the American Medical Association, the American Otological Society, the American Laryngological, Rhinological and Otological Society, and the American Federation of Organizations for the Hard of Hearing:

"Recognizing the fact that the most effective means for prevention of deafness consists in the early detection of hearing impairment, thereby giving opportunity for the prompt removal of contributing causes, and believing it to be one of the important functions of our public school authorities to safeguard the integrity of the special sense organs, as well as the general health of the school child, be it

RESOLVED By the.....that it heartily favors the provision by our public school authorities

for regular periodic examinations of the hearing acuity of all public school children, such examinations to be adequate to detect even slight degrees of hearing loss."

The adoption of this resolution by these national scientific bodies we regard as a most significant endorsement of the work of your committee. Already periodic examinations of the hearing of public school children are being initiated in several of our cities, and medical men and educators are manifesting a new interest on the subject of conserving the hearing.

Your committee feels that this work is still in its infancy, and much remains to be done in the way of research, publicity and, ultimately, legislation, which will insure to every school child in our country a regular periodic examination of his hearing acuity, with the object of preventing possible later hearing loss of a serious degree.

We, therefore, again bespeak the interest and active cooperation of every Fellow of the Academy in this constructive work which you have committed to us. In order that we may be in line with other societies, we recommend the adoption at this meeting of the resolution presented in this report.

Respectfully submitted,

HOWARD V. DUTROW
HAROLD HAYS
CHARLES H. RICHARDSON
GEORGE L. TOBEY
CLAUDE T. UREN
HORACE NEWHART, CHAIRMAN.

The following resolution was adopted:

Recognizing the fact that the most effective means for the prevention of deafness consists in the early detection of hearing impairment, thereby giving opportunity for the prompt removal of contributing causes, and believing it to be one of the important functions of our public school authorities to safeguard the integrity of the special sense organs, as well as the general health of the school child; be it

RESOLVED by the American Academy of Ophthalmology and Oto-Laryngology, that it heartily favors the provision by our public school authorities for regular periodic examinations of the hearing acuity of all public school children, such examinations to be adequate to detect even slight degrees of hearing loss.

REPORT OF COMMITTEE ON NECROLOGY.

The following members have died during the last year:

Dr. Alexander Duane.....New York City
Dr. Marcus Feingold.....New Orleans, La.
Dr. Lee M. Francis.....Buffalo, N. Y.
Dr. James M. Guthrie.....Meridian, Miss.
Dr. Edward P. Hall.....Kansas City, Mo.
Dr. George F. Keiper.....Lafayette, Ind
Dr. Nils E. Remmen.....Chicago, Ill.
Dr. Otis D. Stickney.....Atlantic City, N. J.
Dr. George O. Suffa.....Boston, Mass.
Dr. Oliver Tydings.....Chicago, Ill.
Dr. S. Lewis Ziegler.....Philadelphia, Pa.

DR. LUTHER C. PETER.

Dr. Marcus Feingold.

DR. CHARLES BAHN, New Orleans, La.:

On December 25, 1925, our friend and comrade, Marcus Feingold, fell into that dreamless sleep from which the mortal body does not awaken. Altho fate has deprived us of his companionship, his kindly counsel and memory will always be an inspiration to those who knew him. He lived and loved ophthalmology. His heart of hearts lay in the fields of ocular pathology and of teaching, in which he was second to but few in this country. As an ophthalmic surgeon, he was probably the most technically perfect operator in the South—conservative and accurate. Dr. Feingold was probably one of the most widely read ophthalmologists in this country. He not only spoke and read six languages fluently but had collected a library consisting of almost every ophthalmic publication ever written in German, French and English.

Dr. Feingold was born in Roumania on July 14, 1871, where he received his early education. His undergraduate medical training was received at the University of Vienna, from which he graduated in 1895. He came to America in 1896, and entered into general practice in New Orleans. Shortly afterwards, he confined his practice to eye, ear, nose and throat diseases, and within a very few years to diseases of the eye alone. He built up the very large eye clinic at

the Touro Infirmary from nothing to one of the largest in the South, which may practically also be said of his service at the Charity Hospital at New Orleans. In 1906, Dr. Feingold was tendered the Professorship of Ophthalmology in the undergraduate department of Tulane University, which he filled with the greatest honor and credit to the institution. Dr. Feingold was a member of the American Board of Ophthalmic Examiners, the American Academy of Ophthalmology and Oto-Laryngology, the American Ophthalmological Society, a collaborator in the American Journal of Ophthalmology and the Ophthalmic Year Book, as well as a member of numerous other professional organizations.

The scientific honors which were bestowed on Dr. Feingold reflect in only a small way his sterling qualities, his broad knowledge and his almost uncanny judgment. His achievements will probably not be duplicated in our generation, because they represent the best of European heredity, training and principles transplanted and developed in American institutions. When one recalls that he did not spend practically one well day in the last twelve years, the amount of work that he accomplished is amazing. Aside from teaching and professional meetings of one sort and another, to say nothing of an enormous amount of professional reading and writing, he routinely saw between 150 and 200 patients daily in clinic and office. Over half of his time, and by far the greater part of his energies, was given without financial reward to clinical work and teaching.

I am reminded of the old family physician, who, after having served his community long, well and faithfully, had passed into the great beyond. His friends, in search of the most appropriate token to place at the head of his grave, selected the old worn office sign which read, "Office Upstairs."

Dr. Lee Masten Francis.

Dr. Thomas E. Carmody, Denver, Colorado:

While ophthalmology has sustained a great loss in the last year, the American Academy has sustained a greater loss in our friend, and I am glad to say my friend, Dr. Lee Masten Francis. Cut off in his youth—only forty-eight years of age—he had only begun to live, only begun to practice.

A Pennsylvanian by birth, he was educated at Rush Medical College, graduating in 1891, and serving his internship at the Minnequa Hospital, at Pueblo, Colorado, where he became acquainted with his wife, who added much to his success in life. His family was his treasure—his wife and children and his home life were supreme. Having visited in the family, I feel I never knew a family in which there was more love than in that of Dr. Francis.

Dr. Francis served this Society so well, that we feel his loss very keenly. He served as Secretary from 1911 to 1918, when he was called into the Army and served his country worthily. Upon his return, he was elected President of the American Academy, and served in that capacity in 1919-1920. Besides being a member of this Academy, he was a very efficient operator and enjoyed a large ophthalmic practice. He was a member of the American Ophthalmological Society, of the Buffalo Ophthalmic Club, and at the time of his death was Assistant Professor of Ophthalmology in the medical department of Buffalo University. He was also a member of the staff in several hospitals in Buffalo, where he was loved by the patients, especially in the General Hospital, where he served them freely.

Dr. Francis died of cerebral hemorrhage on April 22, 1926, during the meeting of the American Medical Association in Dallas, where he had read a paper the day previous. In his death, we have lost not only an efficient ophthalmologist and prolific writer, but a valued member of the American Academy of Ophthalmology and Oto-Laryngology.

Dr. George F. Keiper.

Dr. John F. Barnhill, Indianapolis, Ind.:

A large number of the members present will recall Dr. George F. Keiper of Indiana. He was not connected with any medical institution as a teacher, and I mention that fact, for the reason that it is a splendid thing, that a man who was not driven to do so by teaching, still kept up with medicine as did Dr. Keiper.

Dr. Keiper was an Indiana man, out and out. He was born there, he was educated at Depauw University, and he received his medical education in Michigan University. He was connected with universities all his life in some way, not as a teacher, but as a part of the governing body. He was

one of the Board of Trustees of Depauw University, he lived in a university town, Lafayette, the seat of Purdue University, so that he was concerned all his life with educational facilities and educating people.

Tho Dr. Keiper was strictly a Hoosier, his thought was not at all limited to that state. He was very broad minded in medicine. He read more than one language, and he collected from the literature of these languages a great deal that was helpful to him and to his patients, and to the writings which he did. Dr. Keiper was a rather prolific writer, and was the author of a large number of papers, some of which have been before this Academy. He was a member of this Academy he was a member of the Triological Society and other associations. He was at one time President of the Indiana State Medical Association. He loved politics, and in that way took very active interest in all the things that help to push medicine along.

Dr. Keiper died very much as did Dr. Francis. He was found dead in his bed one morning, and it was not known that he had been ill at any time before. It was presumed he died of a heart affection.

Dr. Keiper was very much interested in medicine—in his private practice, his home practice, and the practice in the neighborhood and the community. He did a large charity work and was not of a grasping nature in monetary affairs—rather would he do a thing gratuitously than for money.

Indiana misses Dr. George F. Keiper, this Academy will miss him, but scientific medicine will miss him very greatly.

Dr. Oliver Tydings.

Dr. Joseph C. Beck, Chicago, Illinois:

Dr. Oliver Tydings came to Chicago and began his studies very late in life. At a period when men are about to retire, he first came from Piqua, Ohio, and took up the study of the specialty of otolaryngology. He was a student in my classes, and I was astonished at the interest he showed in perfecting himself in the finer points of the study of otolaryngology. He was handicapped by having been in general practice in a smaller town for a good many years, but in a very short time after his study, as you will recollect, in the early days of the dissection operation of tonsils, he per-

fected an instrument known as the Tydings' Snare, which was much used in this country by many operators, and he was an excellent operator in that line.

Almost immediately after this very brief development in otolaryngology, Dr. Tydings began to teach in the Post-Graduate School, and taught there several times a week in eye, ear, nose and throat diseases. He enjoyed a very good practice in the short time he was in Chicago, and was well liked by his confreres. He wrote a number of papers, particularly on the resection of the nasal septum.

In the later years, he became interested in a hospital in Chicago, in which he took an active part. We were all much shocked to learn of his death.

Dr. S. Lewis Ziegler

Dr. Edward B. Heckel, Pittsburgh, Pa.:

For the privilege of having known Dr. S. Lewis Ziegler for more than a quarter of a century, I am grateful. He was a learned and cultured gentleman. He received his early education at Bicknell College, in Pennsylvania. His medical education was begun at the University of Pennsylvania, supplemented later by studies abroad, and frequent trips abroad. He was a man of very high ideals, a typical physician in many ways. He gave a great part of his life to the Wills Eye Hospital in Philadelphia, and to other hospitals in that city. He was not only interested in medicine itself, but in the human side of it. He gave a great deal of his time to medical associations. He was an active member of the American Medical Association, of the American Ophthalmological Society, and of this Academy.

Dr. Ziegler contributed many articles, and many of his footprints will be followed by the coming generation, because he has left imprints which will last for a long time to come. He was interested in educational institutions, and was a Trustee of his Alma Mater for a number of years. He also had time for civic affairs, and was for one term director of the Department of Public Health in Philadelphia, a position which he filled with distinction.

I feel that while he is gone, we still think of him and may be consoled by the fact that the memory of a well spent life is eternal.

Dr. Harry S. Gradle. I wish to take your time for a moment, to call your attention to a loss which this Academy has sustained, indirectly it is true, but still a loss. Since the meeting in 1906, there has been in attendance at every single meeting of the Academy until this year one whom we now miss. I wish to take this opportunity to pay my respects to the memory of Mrs. Victor Mueller, and I am sure the Academy will join me.

Dr. Thomas E. Carmody. I move that we stand for a moment with bowed heads in memory of our departed members. Carried.

Amendments to Constitution and By-Laws.

Dr. George F. Suker. As the Council has carefully considered these, and as they have been published in the program and have been presented to us this evening, I move that the amendments as proposed by the Senior Member of Council be accepted, and that the Constitution and By-Laws as amended be adopted. Motion seconded by Dr. Berens, and carried. I think I have something quite novel to offer the Academy. Last year the Council appointed a committee, consisting of Dr. Loeb of Chicago, and myself, to consider the advisability of instituting at the John A. Crerar Library, Chicago, a circulating library of eye, ear, nose and throat literature by the members of the Academy. We have had various consultations with the librarian and with members of the Board, and they endorse our opinion that the library shall be called The John A. Crerar Collection of the American Academy of Ophthalmology and Oto-Laryngology. The library of eye, ear, nose and throat literature consists of about 20,000 volumes, which will be open to any member of the Academy who writes for them. Abstracts will be made for members at a nominal cost, and also translations. Any donations the members wish to make to this collection, or to the collection on general medicine, will be received, and book plates will be placed in each volume. The only charge made to the members is the carrying charges to and from the library. There is to be a committee appointed from this Academy to act with one from the John A. Crerar Library, on the advisability of increasing the library. If that is carried out, and I trust the Council members will agree to have it carried out, it will

surely be of great benefit to the members of our specialty thruout the United States.

THE CHAIRMAN. I will ask Dr. Suker to present this at the Thursday morning meeting of the Council.

THE SECRETARY. The following members are recommended by the Council for election:

Anderson, Dr. Carl M., Mayo Clinic, Rochester, Minn.
Beal, Homer A., 1002 Argyle Bldg., Kansas City, Mo.
Daily, Dr. Louis, 647 Kress Bldg., Houston, Texas.
Daily, Dr. Ray Karchmer, 647 Kress Bldg., Houston, Texas.
Ezell, Dr. Herschell C., 288 Lambuth Bldg., Nashville, Tenn.
Fox, Dr. Noah, 5616 S. Michigan Ave., Chicago, Ill.
Gipner, Dr. John F., Mayo Clinic, Rochester, Minn.
Goar, Dr. Everett Logan, 1300 Walker Ave., Houston, Texas.
Hamlin, Dr. Fred Eugene, Shenandoah Life Bldg., Roanoke, Va.
Hands, Dr. Sidney Gillott, 602 1st National Bank Bldg., Davenport, Ia.
Hansel, Dr. French Keller, 1043 Missouri Bldg., St. Louis, Mo.
Harding, Dr. Frederick B., 25 S. 10th St., Allentown, Pa.
Hargitt, Dr. Charles A., 17 Schermerhorn St., Brooklyn, N. Y.
Harrell, Dr. Voss, Holman Bldg., Detroit, Mich.
Hartshorne, Dr. Isaac, 20 W. 59th St., New York City.
Hicks, Dr. Vonnie Monroe, Professional Bldg., Raleigh, N. C.
Jones, Dr. Edmund Lloyd, Wheeling Clinic, Wheeling, W. Va.
Joy, Dr. Harold Henry, 232 Brattle Rd., Syracuse, N. Y.
Lewis, Dr. Eugene R., 1920 Wilshire Bldg., Los Angeles, Cal.
McLaurin, Dr. John G., 1301 Medical Arts Bldg., Dallas, Texas.
McLoone, Dr. John J., 611 Heard Bldg., Phoenix, Ariz.
McMahon, Dr. Bernard John, 604 Missouri Theatre Bldg., St. Louis, Mo.
Maghy, Dr. Charles Alford, Medical Office Bldg., Los Angeles, Calif.
Minsky, Dr. Henry, 215 W. 88th St., New York City.
Morrison, Dr. Wm. Wallace, 39 E. 50th St., New York City.
Myers, Dr. Eugene Lee, 207 Wall Bldg., St. Louis, Mo.
Northcott, Dr. Thomas A., 17 E. 38th St., New York City.
Pember, Dr. Aubrey H., 500 W. Milwaukee St., Janesville, Wis.
Pendexter, Dr. Ralph S., 1424 K St., N. W., Washington, D. C.

Reese, Dr. Monroe David, 3527 Hamilton St., Philadelphia, Pa.
Scal, Dr. Joseph Coleman, 44 W. 74th St., New York City.
Seligstein, Dr. Milton B., 801 Union Planters Bank Bldg., Memphis, Tenn.
Stroud, Dr. Edward Frank, 805 Medical Arts Bldg., Dallas, Texas.
Sweet, Dr. Robert B., 1006 Pacific Southwest Bank Bldg., Long Beach, Calif.
Von der Heydt, Dr. Robert, 25 E. Washington St., Chicago, Ill.
Wagener, Dr. Henry P., Mayo Clinic, Rochester, Minn.
Watkins, Dr. Edwin Dial, 1412 Exchange Bldg., Memphis, Tenn.
Wetherbee, Dr. Charles, Twin Falls, Idaho.
Wright, Dr. Walter Walker, 143 College St., Toronto, Can.
Young, Dr. Charles A., Gill Memorial Eye, Ear & Throat Hospital, 711 S. Jefferson St., Roanoke, Va.

Also the following names from the list not certificated by Boards. Some of the names in the second list have been certificated since this list was printed. These names are as follows:

Brown, Dr. Albert Louis, 2600 Union Central Bldg., Cincinnati, O.
Carmody, Dr. Thomas J., 408 The Temple, Danville, Ill.
Chapman, Dr. Solomon J., 402 Burns Bldg., Colorado Springs, Col.
Cleff, Dr. Oscar, 31 N. State St., Chicago, Ill.
Kettelkamp, Dr. Fred O., Colorado Springs, Col.
McDonald, Dr. Dennis J., 17 E. 38th St., New York City.
Packard, Dr. Louis, Sterling, Col.
Palmer, Dr. Arthur, 2178 Broadway, New York City.
Pentecost, Dr. Reginald S., 90 College St., Toronto, Can.
Reineke, Dr. Geo. F., New Ulm, Minn.
Schulze, Dr. Emile C., Fox Bldg., McKinney, Texas.
Scott, Dr. Merle E., 1st National Bank Bldg., Massillon, O.
Sharp, Dr. Benjamin S., 218½ E. College St., Iowa City, Ia.
Stokes, Dr. Wm. H., 4105 Live Oak St., Dallas, Texas.

Dr. George F. Suker. I move that the Secretary cast the unanimous ballott of the Academy for these members. Motion seconded by Dr. Conrad Berens and carried, and ballot cast.

ELECTION OF OFFICERS.

The Council recommends the following for officers:

President-Elect, Dr. Luther C. Peter, Philadelphia, Pa.
1st Vice-President, Dr. H. W. Woodruff, Joliet, Illinois.
2nd Vice-President, Dr. Ralph Fenton, Portland, Oregon.
3rd Vice-President, Dr. Conrad Berens, New York City.
Treasurer, Dr. Secord H. Large, Cleveland, Ohio.
General Secretary, Dr. W. P. Wherry, Omaha, Nebraska.
Editor of Transactions, Dr. C. Loeb, Chicago, Ill.
Member of Council for 3 years, Dr. Frederick Stauffer, Salt Lake City, Utah.
American Board for Ophthalmic Examinations, Dr. F. P. Calhoun, Atlanta, Ga.

DR. GEORGE F. SUKER. I move that the nominations close and that the Senior Member of the Council cast the unanimous ballot of the Academy for the names read. Motion seconded and carried, and the ballot cast.

DR. LUTHER C. PETER. I hardly know how to express in words my appreciation of the honor which you have given to me by electing me to this Chair. To have been able to serve you for eight years as Secretary was a very great privilege, and inasmuch as you have evidenced your further confidence by electing me to the office of President, I can only say that with the cooperation and counsel of my fellow officers, I promise, in 1928, to the best of my ability to maintain the high standards set by the many men who have preceded me in the presidency of this very distinguished body.

Detroit and Montreal, Canada, were presented as places of meeting. On motion of DR. SUKER, duly seconded, Detroit was selected as the place of meeting for 1927.

DR. HARRY S. GRADLE. A year ago, the Section on Ophthalmology of the American Medical Association adopted the report of the Committee on Compensation for Eye Injuries. Later, this report was adopted by the American Ophthalmological Society, and for the sake of uniformity it would be advisable that the Academy adopt the same report. I, therefore, move that the American Academy of Ophthalmology and Oto-Laryngology endorse the report of the Committee on Compensation for Eye Injuries as officially adopted by the American Medical Association. Motion seconded by DR. CHARLES BAHN, and carried.

THE PRESIDENT. I will appoint as an Auditing Committee: Dr. George W. Jean and Dr. William H. Luedde.

DR. GEORGE F. SUKER. I move that the members of the Academy give a rising vote of thanks to the Local Committee on Arrangements for the manner in which they have taken care of us, and the efforts they have put forth to give us a royal good time. Motion seconded and unanimously carried.

No further business appearing, the Business Session adjourned.

September 16, 1926.

The SECRETARY read four additional names that have been certificated for membership:

Birsner, Dr. Louis J., 25 1st National Bank Bldg., Belleville, Ill.

Cowley, Dr. Robert Henry, Berea, Ky.

Sheahan, Dr. William L., 59 College St., New Haven, Conn.

Neher, Dr. Edwin Manson, Boston Bldg., Salt Lake City.

On motion, duly seconded, these men were elected to membership.

THE PRESIDENT. I will oppoint the following committees:

PUBLICITY, SERVICE AND LEGISLATION. Arthur J. Bedell, John J. Shea.

COMMITTEE ON OPTOMETRY. William H. Wilder, John M. Patton, Charles Bahn.

THE PRESIDENT. At the request of Dr. Crisp, the Editor of the Ophthalmic Year Book, I will appoint a committee to meet with similar committees from other ophthalmologic societies, to consider the question of the future of the Year Book. To represent the Academy I will appoint Dr. Harry S. Gradle. We will now have a supplementary report from the council.

DR. THOMAS E. CARMODY. Some suggestions have come to the Council, that we should hold a meeting at some time on board a boat, making a trip from some port in the United States to some other place, say a cruise on the Carribean Sea. The Raymond Whitcomb Company would be able to take good care of us, but the Council does not feel this the time to suggest it. We simply report it to the membership, and you can think about it, and at some later time which might be suggested, we might actually hold such a meeting. We feel this has been a small meeting, and there is a possibility that that would be a smaller one. However, if a

great many people think favorably of it and desire it, there is a possibility that we might have a meeting that would be very profitable to all of us. We might go to Havana and have the meeting there, and go back and forth in the boat. That is simply a suggestion.

At the Council meeting this morning the President appointed as Secretaries, according to the By-Laws:

John L. Myers, Kansas City, Mo., Oto-Laryngology.

William L. Benedict, Rochester, Minn., Ophthalmology.

Harry S. Cradle, Chicago, Illinois, Graduate Work.

The Council also wishes to propose for honorary membership Mr. Basil Graves of London, our guest of honor at this meeting.

Dr. Edward B. Heckel, Pittsburgh, Pa. I move that Mr. Basil Graves of London be elected to honorary membership of the Academy, the Secretary to cast the ballot. Motion seconded and carried, and ballot cast.

Report of Auditing Committee.

Dr. William H. Luedde. The Auditing Committee has carefully investigated the report of the Treasurer, and finds same to be a model of accuracy, industry and sobriety. On motion this report was accepted as presented.

No further business appearing, the session adjourned.

In order that the Directory may be accurate and up to date, each Fellow is urged to check up on his address, specialty and date of entrance into the Academy, and to report to the General Secretary, Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, Neb., any error or omission. The Transactions are sent to the addresses which appear in the Directory, and if these are incorrect, delay in receiving them will result.

ALPHABETIC LIST

Honorary Fellows

DUNDAS-GRANT, SIR JAMES...148 Harley St., W. 1, London, Eng.
ELLIOT, LT.-COL. R. H.,
54 Welbeck St., Cavendish Sq., London, W., Eng.
ELSCHNIG, PROF. A.,
Ferdinandstrasse, 10, Prague, Czecho-Slovakia
GRAVES, BASIL.....35 Weymouth St., W., London, Eng.
GRAY ALBERT A.....Glasgow, Scotland
JOHNSON, LINDSAY.....Britannia Bldg., West St., Durban, Natal
KIRKPATRICK, LT.-COL. HENRY,
54 Welbeck St., W. I., London, Eng
LAWFORD, J. B., 27 Weymouth St., Portland Pl., W., London, Eng.
LISTER, SIR WILLIAM T., 24 Devonshire Place, W. 1, London, Eng.
NAGER, PROF.F.....Freiestrasse 20, Zurich, Switzerland
VAN DER HOEVE, PROF. J...Rijnsburgerweg, 6 A, Leiden, Holland

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Life Members

BROWN, EDWARD J.(1911)
900 Nicollet Ave., Minneapolis, Minn., OALR
*DAYTON, W. L.(1896)...302 Funke Bldg., Lincoln, Neb., OALR
*FOSTER, HAL(1894)
1014 Chambers Bldg., Kansas City, Mo., ALR
HOWE, LUCIEN.....520 Delaware Ave., Buffalo, N. Y., Op.
MAYER, EMIL.....40 E. 41st St., New York City, N. Y., ALR
STANDISH, MYLES,
51 Hereford St., Back Bay, Boston, Mass., Op.
STEWART, T. M.....901 Union Trust Bldg., Cincinnati, O., OALR

Fellows

A

ABBOTT, W. J.(1919)....8314 Euclid Ave., Cleveland, O., OALR
ADAMS, A. L.(1905)..323 West State St., Jacksonville, Ill., OALR
ADAMS, C.....52 West State St., Trenton, N. J., OALR
ADAMS, DANIEL S.,
520 Hume-Mansur Bldg., Indianapolis, Ind., ALR
ADAMS, ELDRIDGE, S.(1920)
Moore Bldg., San Antonio, Tex.,OALR
ADAMS, FRANK M.(1924)
122 Waterman St., Providence, R. I., ALR

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*Attended 1926 meeting. Numbers in parentheses indicate year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Secretary.

ADAMS, FRED'K. W. (1920) Roosevelt Clinic, Seattle, Wash., OALR
*AGNEW, FRED'K. F. (1920)
120 3rd Ave., S. E., Independence, Ia., OALR
AINSLEE, GEORGE (1921)
606 Oregonian Bldg., Portland, Ore., OALR
ALBRIGHT, LT. P. M., M. C. U. S. Navy, (1922)
U. S. Naval Hosp., Brooklyn, N. Y., ALR
*ALDEN, ARTHUR M. (1922)
537 Frisco Bldg., St. Louis, Mo., ALR
ALDERDYCE, W. W. (1908).....Nasby Blk., Toledo, O., OALR
ALKIRE, H. L.....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS. E. (1920)
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. H. (1916).....Mills Bldg., Topeka, Kan., OALR
ALLEN, G. V. (1921).....835 Kansas Ave., Topeka, Kan., OALR
*ALLEN, J. H.....435 Majestic Bldg., Denver, Colo., ALR
ALMOUR, RALPH (1925) .51 W. 73rd St., New York, N. Y., ALR
ALTER, F. W.....Toledo Medical Bldg., Toledo, O., OALR
ALTRINGER, ARTHUR N.,
1100 Rialto Bldg., Kansas City, Mo., ALR
ALWAY, ROBERT D...423 S. Lincoln St., Aberdeen, S. D., OALR
AMBERG, EMIL.....David Whitney Bldg., Detroit, Mich., ALR
*ANDERSON, CARL M. (1926) Mayo Clinic, Rochester, Minn.,
ANDERSON, W. B.....507 Main Ave., Brownwood, Tex., OALR
*ANDREWS, A. H. (1896) 30 N. Michigan Ave., Chicago, Ill., OALR
ANDREWS, B. F. (1912) 25 E. Washington St., Chicago, Ill., OALR
ANDREWS, H. D.....23 Allen St., Buffalo, N. Y., Op.
ANDREWS, M. P. (1915) Dempsey Bldg., Manitowoc, Wis., OALR
ANNON, WALTER T.....259 S. 17th St., Philadelphia, Pa., ALR
APPLEMAN L. F. (1913)
308 South 16th St., Philadelphia, Pa., Op.
ARBUCKLE, M. F....University Club Bldg., St. Louis, Mo., ALR
ARNOLD, F. J.....182 Pearl St., Burlington, Vt., OALR
ARRELL, WILLIAM.....317 Main, E., Hamilton, Ontario, OALR
ARROWSMITH, H.....170 Clinton St., Brooklyn, N. Y., ALR
ASH, GARRETT G.....1 Main St., Bradford, Pa., OALR
ASHBURY, MARY K.....707 Race St., Cincinnati, O., Op.
ASHLEY, T. W.....466 Park Ave., Kenosha, Wis., OALR
ATKINSON, D. T...1515 Russell Bldg., San Antonio, Tex., OALR
ATKINSON, WALTER SYDNEY (1925)
168 Sterling St., Watertown, N. Y. Op.
AUERBACH, J.....120 W. 86th St., New York City, N. Y., ALR
AYNESWORTH, H. T. (1912)..Amicable Bldg., Waco, Tex., OALR

B

BABBITT, JAMES A. (1924)
2117 Chestnut St., Philadelphia, Pa., ALR
*BAHN, CHAS. A. (1922)
722 Audubon Bldg., New Orleans, La., Op.
BAILEY, F. W. (1912)...Security Bldg., Cedar Rapids, Ia., OALR
BAILEY, H.....840 Landers Bldg., Springfield, Mo., OALR

BAIRD, CHAS. G. (1920).....Beatrice, Neb., OALR
BAKER, C. H.....Crapo Blk., Bay City, Mich., OALR
BAKER, H. B. (1916).....59 Main St., Taunton, Mass., OALR
BALDWIN, KATE W.....1117 Spruce St., Philadelphia, Pa., ALR
BALENTINE, P. L. (1915)
1524 Chestnut St., Philadelphia, Pa., Op.
BALL, J. MOORES.....4500 Olive St., St. Louis, Mo., Op.
BALL, M. V.....214 Penn Ave., W., Warren, Pa., OALR
*BALLENGER, H. C. (1915) 1340 Asbury Ave., Winnetka, Ill., ALR
BALLIN, M. J.....108 E. 60th St., New York City, N. Y., ALR
BALLON, DAVID H. (1924)
170 Crescent St., Montreal, Canada, ALR
*BANE, WM. M.....1005 Republic Bldg., Denver, Colo., OALR
*BANE, W. C.....1005 Republic Bldg., Denver, Colo., OALR
BANISTER, J. M., Bankers Reserve Life Bldg., Omaha, Neb., OALR
BARBER, F. (1913)....75 S. Fitzhugh St., Rochester, N. Y., Op.
BARCK, C.....Humboldt Bldg., St. Louis, Mo., Op.A
BARDENHEIER, F. G. A.....Frisco Bldg., St. Louis, Mo., ALR
BARKAN, A.....Credit Swisse, Zürich, Switzerland, OALR
BARKAN, H.....Medico-Dental Bldg., San Francisco, Cal., Op.
BARKER, CHAS. B.,
224½ W. Oklahoma Ave., Guthrie, Okla., OALR
BARKER, O. G. A. (1909)
Johnstown Trust Co., Johnstown, Pa., Op.
BARLOW, ROY A.....Madison, Wis., ALR
BARNES, L. E. (1914) 6860 South Halsted St., Chicago, Ill., OALR
*BARNHILL, J. F.....Pennway Bldg., Indianapolis, Ind., ALR
BARTHOLOMEW, A. C. (1904)
122 E. Washington St., Ft. Wayne, Ind., OALR
*BASS, A. L. (1924)....738 Francis Bldg., Louisville, Ky., OALR
*BAUM, H. L. (1917)....510 Republic Bldg., Denver, Colo., ALR
BEACH, S. J. (1914).....704 Congress St., Portland, Me., Op.
BEAL, HOMER A. (1926)
1002 Argyle Bldg., Kansas City, Mo.
*BEARD, H. J. (1924)
510 Dollar Bank Bldg., Youngstown, O., OALR
BEATTIE, ROBERT...David Whitney Bldg., Detroit, Mich., OALR
BEATTIE, W. HENRY.....252 Genesee St., Utica, N. Y., OALR
BEATTY, H. G.....150 E. Broad St., Columbus, O., OALR
BECK, F. L. (1920).....408 Hynds Bldg., Cheyenne, Wyo., OALR
*BECK, J. C. (1901).....2551 N. Clark St., Chicago, Ill., ALR
BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*BEDELL, A. J.....344 State St., Albany, N. Y., Op.
*BEEBE, C. S.....509 Wells Bldg., Milwaukee, Wis., Op.
BEEM, I. F.....31 N. State St., Chicago, Ill., OALR
BEGGS, W. F.....2 Lombardy St., Albany, N. Y., OALR
BEIL, J. WALLACE (1918)
316 Argyle Bldg., Kansas City, Mo., OALR
BELL, GEO. HUSTON (1920)
40 E. 41st St., New York City, N. Y., Op.

BELL, M. H. (1913)
First Nat'l Bank Bldg., Vicksburg, Miss., OALR
BELLIN, JOSEPH.....1026 W. 6th St., Los Angeles, Cal., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Kansas City, Mo., Op.
*BENEDICT, W. L. (1919)....Mayo Clinic, Rochester, Minn., Op.
BENNETT, A. G.....26 Allen St., Buffalo, N. Y., Op.
BENNETTO, FREDRICK R.,
172 Woolwich St., Guelph, Ont., Canada, OALR
BENTLEY, NEIL, 1161 David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD (1924)
30 E. 40th St., New York City, N. Y., Op.
BERGERON, J. Z.....104 S. Michigan Blvd., Chicago, Ill., ALR
BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
BERNSTEIN, E. J.....Professional Bldg., Detroit, Mich., OALR
BERRISFORD, PAUL D....812 Lowry Bldg., St. Paul, Minn., OALR
BERRY, D. F.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
BETTISON, D. L.....502 Wilson Bldg., Dallas, Tex., OALR
BEYER, ARTHUR G. (1925)..19 Garfield Pl., Cincinnati, O., OALR
BEYER, LOUIS J. (1918) 449 Delaware Ave., Buffalo, N. Y., ALR
BINGER, H. E.....1027 Lowry Bldg., St. Paul, Minn., OALR
BINKLEY, R. S. (1915)
550 Fidelity Medical Bldg., Dayton, O., OALR
BIRKETT, H. S. (1907) 252 Mountain St., Montreal, Canada, ALR
BIRSNER, L. J. (1926).....1st Nat'l Bk. Bldg., Belleville, Ill.
BLAAUW, E. E.....190 Ashland Ave., Buffalo, N. Y., OALR
*BLACK, M. (1904)...424 Metropolitan Bldg., Denver, Colo., Op.
BLACK, N. M.....Wells Bldg., Milwaukee, Wis., Op.
BLACK, W. D. (1900)..Metropolitan Bldg., St. Louis, Mo., OALR
BLACKBURN, W. J.....661 Reibold Bldg., Dayton, O., OALR
BLAIR, W. W.....Diamond Bank Bldg., Pittsburgh, Pa., Op.
BLAKE, W. F.....516 Sutter St., San Francisco, Cal., Op.
*BLASSINGAME, CHARLES D. (1924)
20 So. Dunlap St., Memphis, Tenn., ALR
*BLEDSOE, R. W. (1908) 1005 Madison Ave., Covington, Ky., OALR
BLICKENSTAFF, A. J. (1922)
804 Peoria Life Bldg., Peoria, Ill., OALR
BLISS, C. B.....411 Columbus Ave., Sandusky, O., OALR
BLUE, J. B. (1916).....Exchange Bldg., Memphis, Tenn., OALR
BLUM, H. N....Maison Blanche Bldg., New Orleans, La., Op.
BOBB, E. V.....1st Nat'l. Bank Bldg., Mitchell, S. D., OALR
BOECKMANN, EGIL.....Lowry Bldg., St. Paul, Minn., OALR
BOEHRINGER, H.W.,
1014 Cooperstown Rd., Upper Darby, Pa., Op.
BOERNER, MORRIS H. (1923)
Scarborough Bldg., Austin, Tex., OALR
BOILER, W. F.....426 S. Dodge St., Iowa City, Ia., OALR
BONE, H. D.....29 Parker St., Gardner, Mass., OALR
BOONE, J. C. (1921)
213 Pythian Bldg., South Bend, Ind., OALR

BOONE, JAMES L.(1924)
N. Y. Eye and Ear Inf., New York City, N. Y., ALR
BOOT, G. W.(1909).....800 Davis St., Evanston, Ill., OALR
*BORDLEY, JAMES, JR...Professional Bldg., Baltimore, Md., Op.
BOUCHER, R. B.....414 Banks Bldg., Vancouver, B. C., OALR
BOURBON, OLIVER P.(1920)
607 So. Hill St., Los Angeles, Cal., OALR
BOUSQUET, J. P. E.(1924)
26 Sherbrook St., W., Montreal, Can., OALR
*BOUVY, HARRY M.(1920) 800 Pittock Blk., Portland, Ore., OALR
BOYCE, G. H.(1912)..408 E. D. St., Iron Mountain, Mich., OALR
BOYCE, WM. B.(1912)
1019 Ludington St., Escanaba, Mich., OALR
BOYD, E. T.(1896).....823 Majestic Bldg., Denver, Colo., OALR
BOYD-SNEE, HARRY, 716 L. M. S. Bldg., South Bend, Ind., ALR
BOZER, HERRMANN E., 438 Delaware Ave., Buffalo, N. Y., ALR
BRADFIELD, J. A. L....State Bank Bldg., La Crosse, Wis., OALR
BRASWELL, C. J.(1922).....726 Mayo Bldg., Tulsa, Okla., OALR
BRAUNLIN, WM. H.(1920)
Marion Natl. Bank Bldg., Marion, Ind., OALR
*BRAWLEY, F. E.....30 N. Michigan Blvd., Chicago, Ill., Op.
BRAWLEY, R. V.....Wallace Bldg., Salisbury, N. C., OALR
BRAY, E. R.....1440 Lincoln Ave., St. Paul, Minn., OALR
BRECKENRIDGE, HARRY E.....209 Sixth St., Racine, Wis., OALR
BREHMER, HARRISON L.(1920)
521 Luna Blvd., Albuquerque, N. M., OALR
BREWER, JOHN D.....618 Main St., Dyersburg, Tenn., OALR
BRIBACH, E. J.....212 Simpson Bldg., Atchison, Kan., OALR
BRICKLEY, D. W.....196 W. Center St., Marion, O., OALR
BRIGGS, H. H.....73 Haywood St., Asheville, N. C., OALR
BRIGGS, W. E.....Box 166, Sacramento, Cal., OALR
BRITTON, G. S.....University Blk., Syracuse, N. Y., OALR
BROBST, C.H.....Central Bank Bldg., Peoria, Ill., OALR
BRODRICK, F. W.....511 Lawrence Bldg., Sterling, Ill., OALR
BROOKS, E. B.....1010 Terminal Bldg., Lincoln, Neb., OALR
BROOKS, E. H.(1915)....808 College Ave., Appleton, Wis., OALR
BROSE, L. D.....501 Upper First St., Evansville, Ind., OALR
BROWN, A. L.(1926)
2600 Union Central Bldg., Cincinnati, O.,
BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, E. T.....30 Church St., Burlington, Vt., OALR
BROWN, H. A.(1912)
719 St. Paul Bldg., San Francisco, Cal., Op.
BROWN, J. E.....370 East Town St., Columbus, O., OALR
BROWN, JOHN C.(1920) 632 Lowry Bldg., St. Paul, Minn., OALR
BROWN, J. M.....1136 W. 6th St., Los Angeles, Cal., ALR
*BROWN, L. E.(1917) 1129 2nd Natl. Bank Bldg., Akron, O., ALR
*BROWN, MORROW D.....146 Imperial Bldg., Denver, Colo., ALR
BROWN, MORTIMER G.(1921)
802 University Bldg., Syracuse, N. Y., ALR

BROWN, SAMUEL HORTON (1910)
1901 Mt. Vernon St., Philadelphia, Pa., Op.
BROWN, W. E. . . 8th Ave. and Amity St., Homestead, Pa., OALR
BROWN, W. H.,
Bk. of Montreal Bldg., Edmonton, Alberta, Can., OALR
BRUDER, J. . . . . 230 W. 79th St., New York City, N. Y., OALR
BRUERE, G. E. (1919) . . . . . Journal Bldg., Portland, Ore., OALR
BRUMM, SETH A. (1922)
818 Stock Exchange Bldg., Philadelphia, Pa., ALR
BRUNER, A. B. (1922) . . . . . Guardian Bldg., Cleveland, O., Op.
BRUNER, W. E. . . . . Guardian Bldg., Cleveland, O., Op.
BRUST, H. O. . . . . 720 S. Crouse Ave., Syracuse, N. Y., ALR
BRYANT, A. G. (1908) . . . . . 502 Beacon St., Boston, Mass., ALR
BUCHANAN, NORMAN D. (1925)
479 Water St., Peterborough, Ont., Canada, OALR
BUCKLEY, J. H. . . . . 1st Natl. Bk. Bldg., Ft. Smith, Ark., OALR
BUCKMAN, E. V. (1906)
70 S. Franklin St., Wilkes-Barre, Pa., OALR
BULL, E. L. . . . . 124 E. State St., Ithaca, N. Y., OALR
BULSON, A. E., JR. (1896)
406 W. Berry St., Ft. Wayne, Ind., OALR
BURCH, F. E. . . . . Hamm Bldg., St. Paul, Minn., Op.
BURDICK, AUSTIN F. (1925)
416 Tussing Blk., Lansing, Mich., OALR
BURKE, T. A. . . . . 446 Rose Bldg., Cleveland, O., OALR
BURLESON, J. H. . . Central Trust Bldg., San Antonio, Tex., OALR
BURNS, S. S. (1919) . . . . . Humboldt Bldg., St. Louis, Mo., ALR
BURROUGHS, G. M. (1911) . . . 5 Broad St., Danielson, Conn., Op.
BURTON, F. A. . . . . 404 Watts Bldg., San Diego, Cal., ALR
BUTLER, MARGARET F., 1831 Chestnut St., Philadelphia, Pa., ALR
BUTLER, ROBERT H. (1920)
227 W. Columbus Ave., Bellefontaine, O., ALR
BUTT, M. M. . . . . 2045 Chestnut St., Philadelphia, Pa., Op.
BUVINGER, CHARLES W. (1921)
50 Washington St., East Orange, N. J., OALR
BYRNES, H. F. . . . . 67 Chestnut St., Springfield, Mass., OALR

C

CAHILL, J. W. . . . . 390 Main St., Worcester, Mass., OALR
CALDWELL, R. . . . State Nat'l Bank Bldg., Little Rock, Ark., OALR
CALHOUN, F. P. (1911) . . . . . Candler Bldg., Atlanta, Ga., OALR
CALLFAS, W. F. . . . . 567 Brandeis Bldg., Omaha, Neb., ALR
CAMERON, WILLIAM G.,
1404 National Realty Bldg., Tacoma, Wash., OALR
*CAMP, W. E. (1920)
910 Donaldson Bldg., Minneapolis, Minn., OALR
CAMPBELL, C. H.,
Columbus State Bk. Bldg., Columbus, Neb., OALR
CAMPBELL, D. A. . . . . David Whitney Bldg., Detroit, Mich., OALR
CAMPBELL, D. M. . . . . David Whitney Bldg., Detroit, Mich., OALR

*CAMPBELL, ROBERT A.,
520 LaSalle Bldg., Minneapolis, Minn., OALR
*CAMPBELL, WM. KING..96 Third Ave., Long Branch, N. J., OALR
CAPRON, E. P.....118 Angell St., Providence, R. I., OALR
CARLETON, RALPH (1914) 10 Temple St., Springfield, Mass., Op.
*CARMACK, J. W. (1919)
505 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*CARMODY, T. E. (1904)..Metropolitan Bldg., Denver, Colo., ALR
CARMODY, T. J. (1926).....408 The Temple, Danville, Ill.,
CARNCROSS, HELEN....58 E. Washington St., Chicago, Ill., Op.
CARPENTER, E. R.....708 Medical Arts Bldg., Dallas, Tex., OALR
CARPENTER, E. W.....201 E. North St., Greenville, S. C., ALR
CARR, G. W. (1909)..30 S. Franklin St., Wilkes-Barre, Pa., OALR
CARROLL, F. (1908).....Ely Blk., Cedar Rapids, Ia., OALR
CARROLL, G. G.....614 Main St., West, Rochester, N. Y., OALR
CARSON, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
CARTER, W. W. (1896)
24 E. 48th St., New York City, N. Y., ALR
CARVILL, L. MAUD (1914)
82 Commonwealth Ave., Boston, Mass., Op.
*CARY, E. H.....4712 Lakeside Drive, Dallas, Tex., OALR
*CARY, G. C.....Canon Block, Grand Junction, Colo., OALR
CAVANAUGH, J. A.....7 W. Madison St., Chicago, Ill., ALR
*CAYCE, E. B. (1912)....Hitchcock Bldg., Nashville, Tenn., OALR
*CHADWICK, IRA B. (1920)
Terminal Bldg., Coffeyville, Kan., OALR
*CHAPMAN, S. J. (1925)
402 Burns Bldg., Colorado Springs, Colo., ALR
CHAPMAN, V. A.....Colby-Abbot Bldg., Milwaukee, Wis., OALR
CHAPPELL, RALPH S. (1925)
305 Terminal Bldg., Indianapolis, Ind., ALR
CHARLES, J. W.....Humboldt Bldg., St. Louis, Mo., Op.
CHARLTON, C. C...1924 S. Illinois St., Atlantic City, N. J., ALR
CHASE, E. FRANK (1918) 1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, SUMNER B. (1925) 630 Snell Bldg., Ft. Dodge, Ia., OALR
CHASE, WALTER D. (1924)
230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN...671 Broad St., Newark, N. J., Op.
CHENERY, W. E...377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W...702 Schweiter Bldg., Wichita, Kan., OALR
CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
CHIPMAN, LEVERETT DEV. (1925)
42 Coburg St., St. John, N. B., Canada, OALR
CLARK, F. T.....92 Broad St., Westfield, Mass., OALR
CLARK, H. S.....LaSalle Bldg., Minneapolis, Minn., OALR
CLARK, I. G.....188 E. State St., Columbus, O., OALR
CLARK, J. S. (1914).....67 Stephenson St., Freeport, Ill., OALR
CLARK, L. H. (1915)...37 Monroe Ave., Rochester, N. Y., OALR
CLAY, G. E. (1917).....436 Peachtree St., Atlanta, Ga., Op.
CLEFF, OSCAR (1926).....31 N. State St., Chicago, Ill.

CLERF, LOUIS H.....128 S. 10th St., Philadelphia, Pa., ALR
CLEVENGER, W. F(1905)
Hume-Mansar Bldg., Indianapolis, Ind., ALR
COATES, G. M.....1736 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN.....Masonic Temple, Marshalltown, Ia., OALR
COCHRAN, R. W.(1911) West and 3rd Sts., Madison, Ind., OALR
COFFELT, THEODORE A..Woodruff Bldg., Springfield, Mo., OALR
COGAN, J. E.....Rose Bldg., Cleveland, O., OALR
COHEN, MARTIN, 1 West 85th St., New York City, N. Y., OALR
COHEN, SAMUEL.....1626 Spruce St., Philadelphia, Pa., ALR
COHN, F.....31 East 60th St., New York City, N. Y., ALR
COHN, S.....416 Luke St., Fulton, Ky., OALR
COLLINS, F. W.....Majestic Bldg., Denver, Colo., ALR
COLLINS, J. D.....90 Main St., Northampton, Mass., ALR
COLVER, B. N.(1913)
182 Manchester St., Battle Creek, Mich., OALR
*COMEE, W. C.(1922).....Glenwood Springs, Colo., OALR
CONLON, F. A.....Bay State Bldg., Lawrence, Mass., OALR
CONNOR, CHAS. E.....Hamm Bldg., St. Paul, Minn., OALR
CONNOR, RAY(1905).....1410 Stroh Bldg., Detroit, Mich., OALR
*COOK, W. ALBERT(1924)...506 Palace Bldg., Tulsa, Okla., OALR
*COOPER, C. E.....1516 Cook St., Denver, Colo., ALR
*COOVER, D. H.(1918)
820 Metropolitan Bldg., Denver, Colo., Op.
COPPS, LYMAN A.....Marshfield, Wis., OALR
CORBETT, JEREMIAH J.(1925)
520 Beacon St., Boston, Mass., OALR
*CORSER, J.B.....345 Wyoming Ave., Scranton, Pa., Op.
*COSTEN, J. B.(1925).....313 Wall Bldg., St. Louis, Mo., ALR
COTT, C. C.....60 W. 88th St., New York City, ALR
COTT, DELZON N.(1924)....1001 Main St., Buffalo, N. Y., ALR
COULTER, C. F.....1st Natl. Bk Bldg., Great Falls, Mont., OALR
COWLEY, R. H.(1926).....Berea, Ky.,
COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
*CRAFT, KENNETH L.(1920)
1003 Hume-Mansur Bldg., Indianapolis, Ind., ALR
CRANE, J. WELLINGTON(1921)
128 Third St., Trenton, N. J., ALR
CREIGHTON, W. J.....258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, W. H.(1913) 530 Metropolitan Bldg., Denver, Col., Op.
CROCKETT, R. L.....Oneida, N. Y., Op.
CROMAN, JOSEPH M. JR.(1924)
Washington Apts., Mount Clemens, Mich., OALR
CROSKEY, JOHN WELSH(1905)
Aldine Hotel, Philadelphia, Pa., Op.
CROSS, A. E.(1914).....Slater Bldg., Worcester, Mass., Op.
CROSS, G. H.(1919).....525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R.....30 N. Michigan Blv., Chicago, Ill., OALR
CULBERTSON, L. R.(1898) Masonic Temple, Zanesville, O., OALR

CULLOM, MARVIN McT.(1910)
208 Hitchcock Bldg., Nashville, Tenn., OALR
CUNNINGHAM, ALLAN R.,
260 Barrington St., Halifax, N. S., OALR
CURDY, R.J.....1134 Rialto Bldg., Kansas City, Mo., Op.
CURRAN, EDW. J.(1919)
401 Waldheim Bldg., Kansas City, Mo., Op.
CURRY, G. E.....Westinghouse Bldg., Pittsburgh, Pa., Op.
CURTIN, EUGENE A.(1923)
703 Connell Bldg., Scranton, Pa., OALR
*CURTIS, W. L.....1503 So. 22nd St., Lincoln, Neb., OALR
CUSHMAN, BEULAH(1924)
25 E. Washington St., Chicago, Ill., Op.
CUTLER, FRANKLIN E..1025 Schofield Bldg., Cleveland, O., ALR

D

DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DABNEY, V.(1913)
1633 Connecticut St., Washington, D. C., ALR
DAILEY, GILBERT L.(1920) 713 N. 3rd St., Harrisburg, Pa., OALR
*DAILY, LOUIS(1926).....647 Kress Bldg., Houston, Tex.,
*DAILY, RAY K.(1926).....647 Kress Bldg., Houston, Tex.
DANCY, A. B.....First Natl. Bank Bldg., Jackson, Tenn., OALR
DANIELS, R.L.....Elks Temple, New Bern, N. C., OALR
DARMER, GEORGE A.(1921)
401 Terminal Bldg., Aurora, Ill., OALR
DAVIDSON, HERMAN P.,(1924)
58 E. Washington St., Chicago, Ill., Op.
DAVIES, T. ALEX(1913)
578 Sherbourne St., Toronto, Can., OALR
DAVIS, A. E.....47 E. 57th St., New York City, N. Y., Op.
DAVIS, FRANK C.....Lewistown, Mont., ALR
DAVIS, G. E.....42 W. 77th St., New York City, N. Y., OALR
DAVIS, HARRY B.(1920)
611 Lathrop Bldg., Kansas City, Mo., OALR
*DAVIS, J. E.....Kress Bldg., McAlester, Okla., OALR
DAVIS, J. L.....135 S. 18th St., Philadelphia, Pa., ALR
DAVIS, RALPH F.(1920) 1104 Selling Bldg., Portland, Ore., OALR
DAVIS, THOMAS W.,
O'Hanlon Bldg., Winston-Salem, N. C., OALR
DAVIS, W. THORNWALL,
927 Farragut Sq., Washington, D. C., Op.
DAYTON, G. O.....Lewisohn Bldg., Butte, Mont., OALR
*DEAN, ALFRED, 304 Gilbert Blk. Bldg., Grand Rapids, Mich.,Op.
*DEAN, F. W.(1904)
Council Bluffs Clinic, Council Bluffs, Ia., Op.
DEAN, L. W.....12 S. Clinton St., Iowa City, Ia., OALR
*DEAN, WALTER.....300 Francis Bldg., Louisville, Ky., OALR
DEBOE, M. P.....621 Eaton St., Key West, Fla., OALR

DECHERD, H. B.....Wilson Bldg., Dallas, Tex., OALR
DECKER, J. C.(1918).....Francis Bldg., Sioux City, Ia., OALR
DEFNET, W. A.....62 Adams St., W., Detroit, Mich., ALR
DEICHLER, LYN WALTER,
2028 Chestnut St., Philadelphia, Pa., OALR
DENMAN, IRA O.....425 Ohio Bldg., Toledo, O., OALR
DENNIS, D. N.....221 W. Ninth St., Eria, Pa., Op.A
DENNIS, E. P.(1921).....311 W. 10th St., Erie, Pa., OALR
*DENNIS, F. L
301 Ferguson Bldg., Colorado Springs, Colo., ALR
DENNIS, G. J.(1910)...25 E. Washington St., Chicago, Ill., ALR
DERBY, G. S.....23 Bay State Rd., Boston, Mass., Op.
DETLING, F.....Chapman Bldg., Los Angeles, Cal., OALR
DEWEESE, CLARENCE...154 N. Upper St., Lexington, Ky., OALR
DEWEY, C. H.(1912)
Sternberg General Hospital, Manila, P. I.,
DICKINSON, B. M
5634 Stanton Ave., E. Liberty Sta., Pittsburgh, Pa., ALR
DINTENFASS, HENRY.....1714 Pine St., Philadelphia, Pa., ALR
DIXON, G. S.....40 E. 41st St., New York City, N. Y., R
DIXON, W. E.,
706 1st Natl. Bank Bldg., Oklahoma City, Okla., OALR
DOANE, L. L.(1905).....Reiber Bldg., Butler, Pa., OALR
DODD, O.(1903).....708 Church St., Evanston, Ill., Op.
DOLMAN, PERCIVAL(1919)
490 Post St., San Francisco, Cal., Op.
DONNELL, NEWMAN R.(1917)
Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, W. D.....Boston Bldg., Salt Lake City, Utah., OALR
DONOVAN, J. A.(1902).....Phoenix Bldg., Butte, Mont., OALR
DORENTO, DRED R.,
Merchants Natl. Bank Bldg., Fort Smith Ark., OALR
DORGE, RICHARD I.(1922)
76 Seymour Ave., S. E., Minneapolis, Minn., OALR
DORSEY, J. G.....201 N. Main St., Wichita, Kan., OALR
DOUGLAS, F. A.(1922)...State Bk. Bldg., La Crosse, Wis., OALR
DOW, F. E.....4 West St., Northampton, Mass., Op.
DOWLING, J. I.....116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T.(1915)
Virginia Mason Hosp. Bldg., Seattle, Wash., OALR
*DUANE, J. F.(1916).....418 Jefferson Bldg., Peoria, Ill., OALR
DUDLEY, W. H.(1902) Brockman Bldg., Los Angeles, Cal., OALR
DULANEY, O.....717 Starks Bldg., Louisville, Ky., OALR
DUNLAP, LAWRENCE G.,
Electric Light Bldg., Anaconda, Mont., OALR
DUNN, I. J.....Masonic Temple, Erie, Pa., OALR
DUNN, O. B.....4th and Railroad Sts., Ironton, O.
DUNNINGTON, JOHN H.(1924)
104 E. 40th St., New York City, N. Y., Op.
DUNTLEY, G. S.(1913).....103 So. Side Sq., Macomb, Ill., OALR

DUTROW, H. V. . . 1040 Fidelity Medical Bldg., Dayton, O., OALR
DUTTON, C. K. . . . . Moberly, Mo., OALR
DWIGHT, C. G. . . . . Pioneer Bldg., Madison, Wis., OALR
DWORETZKY, JULIUS. . . . . 7 Law St., Liberty, N. Y., ALR
DYER, CLYDE P. (1920) Metropolitan Bldg., St. Louis, Mo., Op.

E

*EAREL, A. M. . . . . 516 Honeywell Park, Hoopeston, Ill., OALR
EASTON, J. C. (1904) . . . . . Fairbanks Bldg., Springfield, O., OALR
EDSON, R. . . . . 560 Delaware Ave., Buffalo, N. Y., Op.
*EDWARDS, C. J. (1913)
First National Bank Bldg., Vicksburg, Miss., OALR
EDWARDS, S. R. (1918)
111 N. Lafayette St., South Bend, Ind., OALR
EDWARDS, W. A. . . . . 329 Main St., La Crosse, Wis., OALR
ELLEGOOD, J. A. . . . . Equitable Bldg., Wilmington, Del., OALR
*ELLETT, E. C. (1898) . . . Exchange Bldg., Memphis, Tenn., Op.
ELWOOD, CALVIN R. (1922) Spies Bldg., Menominee, Mich., OALR
EMERSON, L. (1906) . . . . Metropolitan Bldg., Orange, N. J., OALR
ENGLE, H. P. . . . . 200 Olive St., Newton, Ia., OALR
ERSNER, MATTHEW S. . . . . 1729 Pine St., Philadelphia, Pa., ALR
ESTABROOK, C. T. . . . . 390 Main St., Worcester, Mass., OALR
EVANS, JOHN N. (1925)
23 Schermerhorn St., Brooklyn, N. Y., Op.
EWING, ARTHUR E. . . Metropolitan Bldg., St. Louis, Mo., Op.
*EZELL, HERSCHELL C. (1926)
288 Lambuth Bldg., Nashville, Tenn.,

F

FAIRBAIRN, JOHN F. . . . 503 Delaware Ave., Buffalo, N. Y., ALR
FAIRCHILD, NORA M. (1922)
Med. Arts Bldg., Omaha, Neb., Op.
FAIRING, JOHN W. (1924)
204 Tribune Bldg., Greensburg, Pa., OALR
*FAITH, THOMAS. . . . . 31 N. State St., Chicago, Ill., OALR
*FALLOWS, H. D. (1916)
Park Hospital Clinic, Mason City, Ia., OALR
FARMER, A. G. . . . . 1040 Fidelity Med. Bldg., Dayton, O., OALR
FARNSLER, H. H. . . . . 1438 Market St., Harrisburg, Pa., OALR
*FARNSWORTH, M. A. . . . . 2436 6th St., Boulder, Colo., OALR
FARRELL, P. J. . . . . 25 E. Washington St., Chicago, Ill., OALR
*FARRINGTON, P. M. (1904)
Exchange Bldg., Memphis, Tenn., ALR
FAWCETT, I. . . . . 75 12th St., Wheeling, W. Va., OALR
FELLOWS, G. G. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
FELT, CARL L.,
N. E. Cor. Chestnut & 20th Sts., Philadelphia, Pa., ALR
*FENTON, RALPH A. (1921)
806 Medical Arts Bldg., Portland, Ore., OALR

*FERGUSON, E. S.(1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR
FERMAN, JOHN W.(1921)..Stahlman Bldg., Charleroi, Pa., OALR
FIELDING, FRED G.(1925) 72 Ridge St., Glens Falls, N. Y., ALR
*FINNOFF, W. C.(1916).....Imperial Bldg., Denver, Colo., Op.
FISCHER, NATHANIEL A.(1925)
623 Union Trust Bldg., Pittsburgh, Pa., ALR
*FISCHER, V. B.....Mercantile Bk. Bldg., Boulder, Colo., OALR
FISHER, CARL..1028 Pacific Mut. Bldg., Los Angeles, Cal., Op.
*FISHER, JAMES A.(1925)
307 Edgemont Drive, Asbury Park, N. J., OALR
FISHER, WM. A.....31 N. State St., Chicago, Ill., OALR
FISKE, G. F.....25 E. Washington St., Chicago, Ill., OALR
*FITZGERALD, JAMES P.(1924)
104 S. Michigan Blvd., Chicago, Ill., Op.
FLAGG, J. D.(1900).....473 Virginia St., Buffalo, N. Y., Op.A
FLATLEY, T. J.....Reliance Bldg., Moline, Ill., OALR
FLOYD, T. W.(1918).....Lehman Bldg., Peoria, Ill., OALR
FLYNN, JAMES A..1616 Eye St., N. W., Washington, D. C., ALR
FOGARTY, CHAS. W....1826 Summit Ave., St. Paul, Minn., OALR
FOLEY, J. W.(1915).....Majestic Bldg., Denver, Colo., OALR
FORBES, H. H.....40 E. 41st St., New York City, N. Y., ALR
FORBES, S. B.....409 Citizens Bk. Bldg., Tampa, Fla., OALR
*FORGRAVE, L. ROBERT(1922).Logan Bldg., St. Joseph, Mo., OALR
FORSYTHE, S. T.(1919) 235 Colonial Arcade, Cleveland, O., OALR
FORT, A. G.....544 W. Flager St., Miami, Fla., OALR
FOSTER, J. H.....1300 Walker Ave., Houston, Tex., ALR
FOSTER, J. M.....Metropolitan Bldg., Denver, Colo., OALR
FOUTS, JOHN D...1110 Fidelity Medical Bldg., Dayton, O., OALR
FOWLER, EARL B.(1925).....31 N. State St., Chicago, Ill., Op.
FOWLER, S. R.....University Bldg., Syracuse, N. Y., ALR
*FOWLER, WILLIAM(1925)
808 Empire Bldg., Detroit, Mich., OALR
FOX, CHAS. H.(1920)...2116 Central Ave., Kearney, Neb., OALR
FOX, L. W.....303 S. 17th St., Philadelphia, Pa., Op.
*FOX, NOAH(1926)....5616 S. Michigan Ave., Chicago, Ill.,
FOX, R. D.....411 Unity Bldg., Bloomington, Ill., OALR
FRANCHERE, FRED'K E.(1920)
4th and Nebraska Sts., Sioux City, Ia., OALR
FRANK, IRA(1908)....104 S. Michigan Blvd., Chicago, Ill., ALR
FRAZER, ROBERT H.(1925)
Battle Creek Sanitarium, Battle Creek, Mich., ALR
FREEDMAN, L. M.(1920)..419 Boylston St., Boston, Mass., ALR
*FRENCH, R. F.(1913)
Masonic Temple Bldg., Marshalltown, Ia., OALR
FREUDENTHAL, W..24 W. 88th St., New York City, N. Y., ALR
FREY, C. L.....529 Vine St., Scranton, Pa., ALR
FRIEDENWALD, H.....1212 Eutaw Pl., Baltimore, Md., Op.A
FRIEDENWALD, JONAS S.(1925)
1212 Eutaw Pl., Baltimore, Md., Op.

FRIEDMAN, D.....44 W. 77th St., New York City, N. Y., ALR
FRINGER, W. R.....Wm. Brown Bldg., Rockford, Ill., Op.
FULKERSON, C. B.,
Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
FULLENWIDER, C. M.....Barnes Bldg., Muskogee, Okla., OALR
FULLER, HARRY B.(1925)....31 N. State St., Chicago, Ill., OALR
*FULLER, T. E.(1915).....803 Pine St., Texarkana, Tex., OALR
*FULTON, JOHN F.(1920).102 Western St., St. Paul, Minn., OALR
FURGASON, A. P.....Richards Blk., Lincoln, Neb., OALR
FURSTENBERG, ALBERT C.(1925)
330 S. State St., Ann Arbor, Mich., ALR
FUST, JOHN H.(1922).....138 W. 9th St., Erie, Pa., OALR

G

GADBOIS, ARTHUR E.(1921)
421 Norfolk Ave., Norfolk, Neb., OALR
GAILEY, WILLIAM W.(1923)
615 Griesheim Bldg., Bloomington, Ill., OALR
GALE, M. JEAN(1919).....Imperial Bldg., Denver, Colo., Op.
*GALLAHER, T. J.....605 California Bldg., Denver, Colo., ALR
GARBER, J. M.....48 Park Ave., Mansfield, O., OALR
GARRAGHAN, E. F.....25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R.(1913).....Strickland Bldg., Roanoke, Va., OALR
GATEWOOD, EMMETTE T.(1925)
Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L....15 E. 48th St., New York City, N. Y., ALR
GAVIN, E. F.....1 S. Sheridan Rd., Waukegan, Ill., OALR
GEIGER, C. W.(1904).....Cobb Bldg., Kankakee, Ill., OALR
GEORGE, H. W.....19 N. Union St., Middletown, Pa., Op.
GIBSON, R. D....Dollar Savings & Trust, Youngstown, O., OALR
GIFFORD, H.....Med. Arts Bldg., Omaha, Neb., Op.A
GIFFORD, SANFORD R.(1919)
Med. Arts Bldg., Omaha, Neb., Op.
GILBERT, F. Y.....148 Park St., Portland, Me., Op.AR
GILL, E. G.(1919).....McBain Bldg., Roanoke, Va., ALR
GILL, G.....Elyria Savings & Trust Bldg., Elyria, O., OALR
GILL, G. BACHE(1923)
1624 Eye St., N. W., Washington, D. C., ALR
GILLETT, WILBUR G.....201 N. Main St., Wichita, Kas., Op.
GILLETTE, DAVID F.(1925)..811 Park St., Syracuse, N. Y., Op.
GILLUM, JOHN R.(1920) 221 S. 6th St., Terre Haute, Ind., OALR
GIPNER, JOHN F.(1926)....Mayo Clinic, Rochester, Minn.
*GITTINS, T. R.(1920).401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, SAMUEL T.(1924).....Pekin, Ill., OALR
GLEASON, E. B.....2033 Chestnut St., Philadelphia, Pa., OALR
GLOGAU, O.....64 E. 91st St., New York City, N. Y., OALR
*GLOSSER, H. H.(1912)....448 Franklin St., Buffalo, N. Y., Op.
GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR

*GOAR, EVERETT L. (1926) . 1300 Walker Ave., Houston, Tex.
GODDARD, H. M. . . . . 1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J. (1911)
1012 N. Charles St., Baltimore, Md., OALR
GOLDING, JOSEPH E. (1925)
170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, HARRY G. . . . . 57 W. 58th St., New York, N. Y., ALR
GOLDSMITH, P. G. . . . . 84 Carlton St., Toronto, Can., ALR
GOLDSTEIN, M. A. . . . 3858 Westminster Pl., St. Louis, Mo., ALR
GOODYEAR, HENRY M. (1924)
3029 Fairfield Ave., Cincinnati, O, ALR
*GORDON, J. S. (1919) 309 Goldsmith Bldg., Milwaukee, Wis., OALR
GORRELL, T. J.,
E. E. Nose & Throat Hospital, Chicago, Heights, Ill., OALR
GOSNEY, CHAS. W. (1920)
6075 Franklin Ave., Los Angeles, Cal., OALR
GOTTLIEB, M. J. 814 West End Ave., New York City, N. Y., ALR
*GOURLEY, G. F. (1908) . 407 Sinclair Bldg., Steubenville, O., OALR
GOUX, R. S. . . . . 545 David Whitney Bldg., Detroit, Mich., OALR
GRACEY, GEORGE F. . . . . 209 State St., Harrisburg, Pa., OALR
*GRADLE, HARRY S. (1911)
58 E. Washington St., Chicago, Ill., Op.
GRAHAM, H. B. . . . . 210 Post St., San Francisco, Cal., ALR
GRANGER, WAYNE B. (1920)
705 Commercial St., Emporia, Kas., OALR
*GRANT, F. E. (1912)
603 Kal. Natl. Bank Bldg., Kalamazoo, Mich., OALR
*GRANT, P. T. (1920)
1407 Grand Rapids Natl. Bk. Bldg., Grand Rapids, Mich., OALR
GREEN, A. S. . . . . 853 Hyde St., San Francisco, Cal., Op.
GREEN, J. JR. . . . . Metropolitan Bldg., St. Louis, Mo., Op.
GREEN, L. D. . . . . 853 Hyde St., San Francisco, Cal., Op.
GREENE, JOS. B. (1923) . . . Haywood Bldg., Asheville, N. C., ALR
GREENWOOD, A. . . . 82 Commonwealth Ave., Boston, Mass., Op.
*GREEVER, BOYD L. (1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B.,
Security Natl. Bank Bldg., Sioux Falls, S. D., OALR
GRIM, U. J. . . . . 30 N. Michigan Blvd., Chicago, Ill., OALR
GRISCOMB, J. MILTON . . 1925 Chestnut St., Philadelphia, Pa., ALR
GROSS, JULIUS E. (1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N. . . . . 378 Dakota St., Huron, S. D., OALR
*GROVE, W. E. (1914) . . . 1330 Wells Bldg., Milwaukee, Wis., ALR
GRUSHLAW, I. . 271 Central Pk., W., New York City, N. Y., ALR
*GSELL, J. F. (1920) . . . . . 911 Beacon Bldg., Wichita, Kas., OALR
GUERINOT, ALBERT J. (1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR
GUGGENHEIM, L. K. . . . . Carleton Bldg., St. Louis, Mo., ALR
GUNDELACH, C. A.,
825 University Club Bldg., St. Louis, Mo., ALR

GUTHRIE, A. L.(1910)
Amer. Natl. Bank Bldg., Oklahoma City, Okla., OALR
GUTTMAN, J....987 Madison Ave., New York City, N. Y., OALR
*GUYTON, B. S.(1918).....Carter Bldg., Oxford, Miss., OALR
GWINN, G. E.(1916)
704 Russell Bldg., San Antonio, Tex., OALR

H

HAAS, FRED'K J.....Masonic Bldg., Leavenworth, Kas., OALR
HACKNEY, F. J...630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, H. C...913 Union Natl. Bk. Bldg., Houston, Tex., OALR
HAGEMAN, JOHN A.(1925) Highland Bldg., Pittsburgh, Pa., ALR
HAGER, W. A.....111 N. Lafayette St., South Bend., Ind., OALR
HAGLER, A. L.....Hagler Bldg., Springfield, Ill., OALR
HAGLER, E. E.(1896).....Hagler Bldg., Springfield, Ill., OALR
HALL, ALICE K.(1924).25 E. Washington St., Chicago, Ill., ALR
*HALL, G. C.(1907).....705 Brown Bldg., Louisville, Ky., OALR
*HALSTED, F. S.....708 Metropolitan Bldg., Denver, Colo., OALR
*HAMLIN, FRED E.(1926)
Shenandoah Life Bldg., Roanoke, Va.
HAMPTON, R. R.....Boston Bldg., Salt Lake City, Utah., OALR
HANCOCK, J. C.(1914)
135 Cambridge Pl., Brooklyn, N. Y., Op.
*HANDS, SIDNEY G.(1926)
602 1st Nat'l Bk. Bldg., Davenport, Ia.
HANEY, WM. P.....Medical Arts Bldg., Omaha, Neb., OALR
HANS, C. L.(1921).....19 W. 7th St., Cincinnati, O., OALR
*HANSEL, F. K.(1926).1043 Missouri Bldg., St. Louis, Mo.
HANSEN, ERLING W.,
910 Donaldson Bldg., Minneapolis, Minn., OALR
HARBERT, J. P.(1918)..135 N. Ulain St., Bellefontaine, O., OALR
HARDESTY, J. F.(1920)
311 Metropolitan Bldg., St. Louis, Mo., Op.
*HARDING, F. B.(1926).....25 S. 10th St., Allentown, Pa.
HARDY, W. F.(1914).....Humboldt Bldg., St. Louis, Mo., Op.
*HARGITT, CHARLES A.(1926)
17 Schermerhorn St., Brooklyn, N. Y.
*HARKNESS, G. F.(1911).509 Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS(1926).....Holman Bldg., Detroit, Mich.
HARRIS, C. M.(1910)
Johnstown Trust Bldg., Johnstown, Pa., OALR
HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
*HARRIS, ROBIN.....Columbian Tower, Memphis, Tenn., ALR
HARRIS, W. C.....Groton Bldg., Cincinnati, O., ALR
HARRISON, W. G.....Empire Bldg., Birmingham, Ala., OALR
HARTLEY, F. A.....Fairbanks Bldg., Springfield, O., OALR
*HARTSHORNE, ISAAC(1926)
20 W. 59th St., New York City, N. Y.
HARTZEL, SOL. M.....Dollar Bank Bldg., Youngstown, O., OALR

HARVEY, N. D.....114 Waterman St., Providence, R. I., OALR
HASKELL, A. D.....145 High St., Portland, Me., Op.
HASKIN, W. H.(1913)...40 E. 41st St., New York, N. Y., ALR
HASPEL, D. M.(1911)
1214 Maison Blanche Bldg., New Orleans, La., OALR
HASTY, FREDERICK E.(1925)
420 Lambuth Bldg., Nashville, Tenn., OALR
HATCH, C. B.(1908).....3 W. Church St., Newark, O., OALR
HAUER, A. M.....327 E. State St., Columbus, O., OALR
HAUGHEY, W.(1911).24 W. Main St., Battle Creek, Mich., OALR
HAWLEY, C. W.....30 N. Michigan Blvd., Chicago, Ill., OALR
HAWMAN, ERLE G...4501 N. Broad St., Philadelphia, Pa., OALR
*HAYDEN, AUSTIN A...25 E. Washington St., Chicago, Ill., OALR
HAYS, H. N.(1912).22 W. 47th St., New York City, N. Y., ALR
HAZLETT, L. R.....117 S. Main St., Butler, Pa., OALR
HEALEY, JOHN F.....503 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F.....Masonic Temple Bldg., Erie, Pa., OALR
HEARD, MARY K.....41 W. Main St., North East, Pa., Op.
HEATH, PARKER(1924)
David Whitney Bldg., Detroit, Mich., Op.
*HECKEL, E. B.(1902)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
HEEB, H. J.....Loan & Trust Bldg., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEGGIE, N. M.(1905)...Buckman Bldg., Jacksonville, Fla., OALR
HEITGER, J. D.(1905)...808 Francis Bldg., Louisville, Ky., OALR
HEMPSTEAD, BERT E.....Mayo Clinic, Rochester, Minn., ALR
HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL.....The Rochambeau, Washington, D. C., Op.
HENNINGER, L. L...205 Citizens Bk. Bldg., Pasadena, Cal., OALR
HENTON, GEO. E.(1918).412 Morgan Bldg., Portland, Ore., OALR
HERBERT, A. W.....119 E. Lehigh Ave., Philadelphia, Pa., OALR
*HERRIMAN, L. L.(1918).Physicians Bldg., Alamosa, Colo., OALR
HESS, W. L.....1554 California St., Denver, Colo., OALR
HETRICK, LLEWELLYN E.,
20 East 53d St., New York City, N. Y., ALR
*HETZEL, C. C.(1906).....512 Eccles Bldg., Ogden, Utah., OALR
*HICKS, VONNIE M.(1926)
Professional Bldg., Raleigh, N. C.
HIGBEE, E. H.(1908).Grand and Olive Sts., St. Louis, Mo., Op.
HIGGINBOTHAM, T.L.,
Solomon Clinic Bldg., Wichita, Kas., OALR
HIGGINS, REUBEN P.(1924)..20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G.....1015 Wells Bldg., Milwaukee, Wis., OALR
HILL, CLARENCE E.(1919)...160 Bloor St., Toronto, Can., Op.
*HILL, EMORY(1916)..Professional Bldg., Richmond, Va., Op.
*HINMAN, EUGENE E.,(1924)
146 Washington Ave., Albany, N. Y., ALR
HITZ, H. B.....Goldsmith Bldg., Milwaukee, Wis., ALR
HODSDON, B. F.....25 N. E. First Ave., Miami, Fla., OALR
HOFFMAN, WALTER F....916 Leary Bldg., Seattle, Wash., OALR

HOFMAN, WILLIAM P.(1925).....Davenport, Ia., OALR
HOGAN, WALTER L.(1924).750 Main St., Hartford, Conn., OALR
HOGSHEAD, J. M....602 Georgia Ave., Chattanooga, Tenn., OALR
HOGUE, D. W.(1908).....Fairbanks Bldg., Springfield, O., OALR
HOHF, J. A.(1915).....Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK,
206 Wilhelm Blk., Traverse City, Mich., OALR
HOLLOWAY, T. B.....1819 Chestnut St., Philadelphia, Pa., Op.
HOLMES, BENJ. H.....432 Baker Blk., Racine, Wis., OALR
HOLT, E. E. JR.....823 Congress St., Portland, Me., Op.A
HOMPES, J. J.....Security Mutual Bldg., Lincoln, Neb., OALR
HOOD, T. C.....Hume-Mansur Bldg., Indianapolis, Ind., Op.
HOPKINS, W. E....515 Park Ave., New York City, N. Y., OALR
HOTCHKISS, W. W....195 Forest Ave., Jamestown, N. Y., OALR
HOURN, GEORGE E.(1920)
703 University Club Bldg., St. Louis, Mo., ALR
HOVDE, A. G.(1916).Board of Trade Bldg., Superior, Wis., OALR
*HOWARD, C. N.(1919).....210 S. Indiana, Warsaw, Ind., OALR
HOWARD, E. F.(1919)
First Natl. Bank Bldg., Vicksburg, Miss., ALR
HOWARD, EDWARD L.....First Natl. Bk., Ft. Worth, Tex., OALR
HOWARD, J. W.....Rialto Bldg., Kansas City, Mo., OALR
HOWARD, LLOYD G.....Council Bluffs, Iowa, OALR
HOWARD, WILLIAM H.,
137 N. Fairview Ave., St. Paul, Minn., OALR
HOWE, ARCHIBALD W.,
Puget Sound Bk. Bldg., Tacoma, Wash., OALR
HUBBARD, A. E.....30 Radcliffe Rd., Buffalo, N. Y., Op.
HUGHES, LEE W.(1925)...1019 Broad St., Newark, N. J., OALR
HUGHES, THOMAS E.(1922)
Medical Arts Bldg., Richmond, Va., ALR
HUGHES, W. F....Hume-Mansur Bldg., Indianapolis, Ind., Op.
HURD, L. M.(1910).39 E. 50th St., New York City, N. Y., ALR
HURFF, J. W.(1919)...86 Washington St., Newark, N. J., OALR
HURLEY, E. D.(1914).....419 Boylston St., Boston, Mass., Op.
HURLEY, E. H.....419 Boylston St., Boston, Mass., Op.
HURST, V. REEVES.....Longview, Tex., OALR
HUSIK, DAVID N.(1921).1610 Spruce St., Philadelphia, Pa., ALR
HUSSEY, E. J.....276 High St., Holyoke, Mass., Op.LR

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IGLAUER, S.....707 Race St., Cincinnati, O., ALR
IMPERATORI, C. J....17 E. 38th St., New York City, N. Y., ALR
INGERMAN, S. M.(1909)
418 Central Park, W., New York City, N. Y., OALR
INGERSOLL, J. M.(1908).....Osborn Bldg., Cleveland, O., ALR
INGLIS, H. J.....43 Bay State Bldg., Boston, Mass., ALR
INGRAM, L. C.(1915).....Clinic Bldg., Olanda, Fla., OALR
IRVIN, EDGAR H.....321 W. Rio Grande St., El Paso, Tex., OALR

IRVINE, A. R.(1918)
709 Westlake Professional Bldg., Los Angeles, Cal., Op.
IRWIN, F. N.....114 E. 54th St., New York City, N. Y., OALR
IRWIN, V. J.....389 Main St., Springfield, Mass., OALR
IRWIN, VINCENT J., JR.(1921)
274 Main St., Springfield, Mass., OALR
ISAACS, D.....Brandeis Theater Bldg., Omaha, Neb., OALR
ISRAEL, J. P...U. S. Veterans Bureau, San Francisco, Cal., OALR
ISRAEL, N. E.....404 Carter Bldg., Houston, Tex., OALR
ISRAEL, S.....Carter Bldg., Houston, Tex., OALR
IVINS, H. M.(1910)...434 Higley Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F.....63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, C.....128 S. 10th St., Philadelphia, Pa., L
*JACKSON, E.(1902).....314 14th St., Denver, Colo., Op.
JACKSON, J. D.(1920).....2324 West 8th St., Erie, Pa.
JACOBS, M. W.....Carleton Bldg., St. Louis, Mo., Op.
JACOBS, S. M.....1018 E. 163rd St., New York City,N.Y., OALR
JACOBY, D. P. A.(1912).....106 Touro St., Newport, R. I., Op.AL
JAMES, J. H.....Natl. City Bank Bldg., Mankato, Minn., OALR
JARECKY, H.....138 W. 86th St., New York City, N. Y., OALR
*JEAN, GEORGE W.....7 East Valerio, Santa Barbara, Cal., Op.
JENNINGS, J. E.....Carleton Bldg., St. Louis, Mo., OALR
*JESSAMAN, L. W.(1914)
132 Union Ave., Framingham, Mass., OALR
JOBSON, G. B.(1912).....4 Printz Bldg., Franklin, Pa., OALR
JOHNSON, A. E.(1919)
Citizens Bank Bldg., Watertown, S. D., OALR
JOHNSON, H. R.....512 Walnut Ave., Fairmont, W. V., OALR
JOHNSON, OTIS H.,
U. S. Veterans' Bureau, Dallas, Texas, OALR
JOHNSON, THOMAS H.,
30 W. 59th St., New York City, N. Y., Op.
*JOHNSTON, W. H.(1912).....Laurel Bldg., Muscatine, Ia., OALR
*JOHNSTON, WILSON 309 Albemarle Terrace, Portland, Ore., OALR
JONES, CHARLES C.(1924)...19 Garfield Pl., Cincinnati, O., ALR
*JONES, E. L.(1913)
First Natl. Bank Bldg., Cumberland, Md., OALR
*JONES, EDMUND L.(1926)
Wheeling Clinic, Wheeling, W. Va.
JONES, E. W.....44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W.....Times Bldg., Alton, Ill., OALR
JONES, LEONARD W.(1925)
53 S. Fitzhugh St., Rochester, N. Y., OALR
JORDAN, G. T.....30 N. Michigan Blvd., Chicago, Ill., OALR
*JOY, HAROLD H.(1926)...232 Brattle Rd., Syracuse, N. Y.,
JOYES, CRITTENTON,
Farmers & Merch. Natl. Bk. Bldg., Ft. Worth, Tex., OALR

JOYNT, ALBERT J. (1924) . . . . . Black Bldg., Waterloo, Iowa, OALR
JUDD, HAROLD B. (1917)
47 E. 57th St., New York City, N. Y., ALR
JUDGE, HARRY V. (1920) . . . . . 46 Willett St., Albany, N. Y., Op.

K

KAHN, HARRY . . . . . 104 S. Michigan Blvd., Chicago, Ill., ALR
KAHN, MYRON E . . . . . 29 E. Madison St., Chicago, Ill., ALR
KAUFFMAN, ARNOLD B. (1922)
305 Gurney Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. S . . . . . 1923 Spruce St., Philadelphia, Pa., ALR
KEARBY, D. O. . 718 Hume-Mansur Bldg., Indianapolis, Ind., ALR
KEELER, J. C . . . . . 254 S. 16th St., Philadelphia, Pa., ALR
KEIM, WILLIAM F. (1924) . 2 Lombardy St., Newark, N. J., OALR
KEITH, D. M . . . . . 414 N. Main St., Rockford, Ill., ALR
*KEITH, W. E . . . . . 807 Waldheim Bldg., Kansas City, Mo., OALR
KELLER, S. A . . . . . Sioux Falls, S. D., OALR
KELLEY, C. W., JR . . . . . 328 Francis Bldg., Louisville, Ky., OALR
KEMLER, JOSEPH J. (1922) . 1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. R . . . . . 614 Osborn Bldg., Cleveland, O., ALR
KENNEY, WM. L . . . . Corby Forsee Bldg., St. Joseph, Mo., OALR
KENNON, WILLIAM G. (1924)
Lambuth Bldg., Nashville, Tenn., OALR
KERRICK, STANLEY E . . 407 Security Bldg., Hollywood, Cal., OALR
KERRISON, P.D . . . . 58 W. 56th St., New York City, N. Y., ALR
KETTELKAMP, FRED O. (1926) . . . . Colorado Springs, Colo.,
KEY, B. W. (1914) 100 W. 59th St., New York City, N. Y., Op.
KEYS, MORTON J . . . 307 Jones Blk., Victoria, B. C., Canada, OALR
*KIEFER, H. A. (1915)
406 Brockman Bldg., Los Angeles, Cal., OALR
KIEHLE, FRED'K A . . . . . 1000 Corbett Bldg., Portland, Ore., OALR
KILBOURNE, PERLEY H. (1924)
Fidelity Med. Bldg., Dayton, O., OALR
*KIMBERLIN, J. W. (1915)
900 Rialto Bldg., Kansas City, Mo., Op.
KINCAID, J. H . . . . 421 W. Church Ave., Knoxville, Tenn., OALR
KING, C. (1910) . . . . . Union Central Bldg., Cincinnati, O., Op.
KING, CHARLES R. (1920) . . . 471 Spitzer Bldg., Toledo, O., OALR
*KING, EDWARD . . . . . 707 Race St., Cincinnati, O., ALR
KING, G. L . . . . . 537 E. Market St., Alliance, O., OALR
KING, J. J . . . . . 40 E. 41st St., New York City, N. Y., ALR
KING, O. H . . . . . Dugan Stuart Bldg., Hot Springs, Ark., OALR
*KIRBY, DANIEL B. (1924)
30 W. 59th St., New York City, N. Y., Op.
KIRKENDALL, J. S . . . . . 315 N. Aurora St., Ithaca, N. Y., OALR
KIRKPATRICK, S . . . . . Lauderdale Ave., Selma, Ala., OALR
KISER, J. D . . . . Central Natl. Bk. Bldg., Bartlesville, Okla., OALR
KISTLER, O. F . . . . . 43 N. Franklin St., Wilkes-Barre, Pa., Op.AR
KISTNER, F. B . . . . . Stevens Bldg., Portland, Ore., OALR

KITTLESON, T. N...101 Lincoln Ave., Fergus Falls, Minn., OALR
*KLINE, H. G.....120 East Washington, Syracuse, N. Y., ALR
KLINEDIENST, J. F.....220 S. George St., York, Pa., OALR
KLINGER, THOS. O.(1920)
314 Landers Bldg., Springfield, Mo., OALR
KNAPP, A.....10 E. 54th St., New York City, N. Y., Op.
KNAPP, B.....Cleveland Life Bldg., Evansville, Ind., OALR
KNAPP, FRANK N.(1925)
309 Bradley Bldg., Duluth, Minn., OALR
*KNEEDLER, G. C.(1912) Professional Bldg., Pittsburgh, Pa., ALR
KNODE, A. R.....Omaha Natl. Bank Bldg., Omaha, Neb., ALR
KOLLOCK, C. W.....86 Wentworth, Charleston, S. C., OALR
KOOBS, HERMAN J. G.(1920)
4th and Walnut Sts., Rogers, Ark., OALR
KOPETZKY, S. J....51 W. 73rd St., New York City, N. Y., ALR
KRAFT, O. H.....25 E. Washington St., Chicago, Ill., Op.
KRAUSS, F.....1701 Chestnut St., Philadelphia, Pa., OALR
KREBS, A.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
KRENTZER, A. G..Trust Natl. Bank Bldg., Milwaukee, Wis., ALR
KRESS, G. H.....245 Bradbury Bldg., Los Angeles, Cal., OALR
KRIEGER, G. M.....119 E. 6th St., Michigan City, Ind., OALR
*KRIEGER, WM. A.(1919)
35 Market St., Poughkeepsie, N. Y., OALR
KRUG, E. F.(1908)..12 W. 44th St., New York City, N. Y., OALR
KRUSKAL, ISAAC D.(1925)
863 Prospect Pl., Brooklyn, N. Y., Op.
KULLY, B. M.....631 City Natl. Bk. Bldg., Omaha, Neb., ALR

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LA FORCE, B. D.....1140 Beverley Rd., Altadena, Cal., OALR
LA FORCE, E. F.....Tama Bldg., Burlington, Ia., OALR
LAMB, F. W.(1908)
209 Provident Bk. Bldg., Cincinnati, O., OALR
LAMB, HARVEY(1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, R. S.....Stoneleigh Ct., Washington, D. C., Op.
*LANCASTER, W. B.(1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDES, GEORGE A.....110½ S. 18th St., Parsons, Kas., OALR
LANDFRIED, C. J.,
607 Land Title & Guarantee Bldg., New Orleans, La., ALR
LANGWORTHY, H. G.....10th & Bluff Sts., Dubuque, Ia., OALR
LANING, J. H.(1918) 726 Lathrop Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M.(1902).Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H.(1904)....2323 Prospect Ave., Cleveland, O., ALR
LARKIN, B. J.(1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.

LARSEN, C. L. (1916) .....Lowry Bldg., St. Paul, Minn., OALR
*LARUE, C. L. (1922) ...17 Physicians Bldg., Boulder, Colo., OALR
LASSALLE, ALBERT.....493 St. Denis St., Montreal, Can., OALR
*LASSEN, F. (1915)...First Natl. Bank Bldg., Pueblo, Colo., ALR
LA VIGNE, A. A.....11 E. 48th St., New York City, N. Y., OALR
LAWRENCE, G. A....Medical Arts Bldg., Philadelphia, Pa., Op.
LEA, JOS. A.....1125 Rialto Bldg., Kansas City, Mo., ALR
LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
*LEDERER, FRANCIS L. (1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W.....369 Broad St., Providence, R. I., OALR
*LEFLER, ANNA B. (1918)
610 S. Broadway, Los Angeles, Cal., OALR
*LEHAN, J. W.....1620 12th Ave., Greeley, Colo., OALR
*LEMERE, H. B. (1912)
330 Bankers Reserve Life, Omaha, Neb., OALR
LEMOINE, A. N. (1920)..Lathrop Bldg., Kansas City, Mo., Op.
LENKER, JOHN N.....Osborn Bldg., Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette St., South Bend., Ind., OALR
LEONARD, ARTHUR C..505 Bryant Bldg., Kansas City, Mo., OALR
LERNER, MACY L. (1923) 332 Park Ave., Rochester, N. Y., Op.
LESTER, H. S.....204 N. Park St., Streator, Ill.,
LEVY, L.....Bank of Commerce Bldg., Memphis, Tenn., OALR
*LEVY, R. (1896).....Metropolitan Bldg., Denver, Colo., ALR
*LEWIS, A. C. (1916).....Exchange Bldg., Memphis, Tenn., OALR
*LEWIS, EUGENE A. (1926)
730 S. Catalina, Los Angeles, Cal.
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
LEWIS, F. P.....454 Franklin St., Buffalo, N. Y., Op.
LEWIS, GEORGE A. (1920)...29 Main St., Roundup, Mont., OALR
LEWIS, J. D.....1534 Estado St., Santa Barbara, Cal., OALR
LEWIS, J. P (1915).....543 Boylston St., Boston, Mass., ALR
LEWIS, WILLIAM W. (1921)
836 Lowry Bldg., St. Paul, Minn., OALR
LIBBY, GEORGE F. (1904)....Majestic Bldg., Denver, Colo., Op.
*LICHTENBERG, J. S...1119 Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, CHAS. J.,
519 Portsmouth Bldg., Kansas City, Kans., OALR
LIFSCHUTZ, JACOB (1925)
25 E. Washington St., Chicago, Ill., OALR
*LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LINN, E. G. (1913).....Fleming Bldg., Des Moines Ia., OALR
LINN, JAY G.....7026 Jenkins Arcade, Pittsburgh, Pa., OALR
LINTLEMAN, F. R. (1917) 312 Hayes Blk., Janesville, Wis., OALR
LISCHKOFF, M. A. (1913)....Blount Bldg., Pensacola, Fla., OALR
*LITCHFIELD, J. T. (1921)
609 Yeats Bldg., Minneapolis, Minn., OALR
LLOYD, A. W. (1915) 504 Hammond Bldg., Hammond, Ind., OALR
LLOYD, RALPH I. (1925).14 Eighth Ave., Brooklyn, N. Y., Op
LOEB, C. (1907).....25 E. Washington St., Chicago, Ill., Op.
LOEB, H. W.....1402 So. Grand Blvd., St. Louis, Mo., ALR

LOFTUS, J. E. (1921)
605 Medical Arts Bldg., Philadelphia, Pa., ALR
LOKEY, H. M. . . . . 41 Doctors Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE (1923)
186 Meserole St., Brooklyn, N. Y., Op.
LOOMIS, E. A. . . . . Donaldson Bldg., Minneapolis, Minn., OALR
LOOPER, EDWARD A. . . . . 605 Park Ave., Baltimore, Md., OALR
LORIE, A. . . . . Commerce Bldg., Kansas City, Mo., ALR
LORING, J. BROWN . . . . . 25 E. Washington St., Chicago, Ill., OALR
LOUCHERY, D. C. . . . . 214 W. Main St., Clarksburg, W. Va., OALR
LOVE, JOHN G. . . . . Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWRY, BLACKBURN W. (1925)
408 Citrus Exch. Bldg., Tampa, Fla., OALR
LUBMAN, MAX. . . . 135 E. 65th St., New York City, N. Y., ALR
*LUEDDE, W. H. (1916) Metropolitan Bldg., St. Louis, Mo., Op.
LUKENS, CHARLES (1906) . . . . 316 Michigan St., Toledo, O., OALR
LUKENS, ROBT. McD.,
1308 Hunting Park Ave., Philadelphia, Pa., ALR
*LUX, PAUL (1925) 409 Commerce Bldg., Kansas City, Mo., ALR
LYMAN, HARRY W. . . . . Carleton Bldg., St. Louis, Mo., ALR
*LYNCH, R. C. (1913)
632 Maison Blanche Bldg., New Orleans, La., ALR
LYONS, HORACE R. . . . . 30 N. Michigan Blvd., Chicago, Ill., ALR

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MCBRIDE, W. O. . . . 2906 S. Fairfield Ave., Ft. Wayne, Ind., OALR
MCCALL, JULIUS W. (1925)
515 Osborn Bldg., Cleveland, O., ALR
MCCANNELL, ARCHIBALD D. . . . . Colcord Blk., Minot, N. D., OALR
*MCCARTNEY, OLIVER P. (1920)
707 Elks Bldg., Kansas City, Kans., OALR
MCCASKEY, C. H.,
Continental Bank Bldg., Indianapolis, Ind., ALR
*MCCAW, JOHN A. . . . . 418 Majestic Bldg., Denver, Colo., Op
MCCLELLAND, CARL C.,
David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E. . . . . Alter Bldg., New Kensington, Pa., OALR
*MCCOOL, J. L. (1911) 725 Sherwood Drive, Portland, Ore., Op.,
MCCOY, J. . . . . 750 Fifth Ave., New York City, N. Y., ALR
MCCREADY, J. H. . . . . Empire Bldg., Pittsburgh, Pa., ALR
MCCULLOCH, R. J. P. . . . . 102 College St., Toronto, Can., OALR
MCDERMOTT, JOHN J. (1924)
St. Joseph Sanitarium, St. Joseph, Mich., OALR
*MCDIARMID, H. O. (1918)
Clement Block, Brandon, Manitoba, Can., OALR

McDONALD, C. L. . . . . 432 Ainsfield Bldg., Cleveland, O., OALR
McDONALD, DENNIS. . 17 E. 38th St., New York City, N. Y.
McDONALD, DENNIS J. (1926)
17 E. 38th St., New York City, N. Y.
McDOWELL, N. D. . . . 275 Alexander St., Rochester, N. Y., OALR
McFARLANE, MURRAY. . . . 190 Bloor St., E., Toronto, Can., OALR
McGINNIS, EDWIN. . . 104 S. Michigan Blvd., Chicago, Ill., ALR
McGIVERN, CHAS. S. 805 Pacific Ave., Atlantic City, N. J., ALR
McGUIRE, H. H. . . . 105 N. Braddock St., Winchester, Va., OALR
*McHENRY, D. D. (1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McKEE, C. W. . . . . Coulter Bldg., Greensburgh, Pa., OALR
McKEE, GEORGE J.,
1118 Westinghouse Bldg., Pittsburgh, Pa., ALR
*McKEE, S. H. (1910) . . . . 158 Crescent St., Montreal, Can., Op.
McKELVEY, A. D. M. . . . . 160 Bloor St., Toronto, Can., ALR
McKENNAN, J. W
Washington Trust Bldg., Washington, Pa., Op.
McKEOWN, E. E. . . . . Metropolitan Bldg., Denver, Colo., OALR
McKINNEY, ALEXANDER R.,
Bearinger Bldg., Saginaw, Mich., OALR
McKINNEY, R. . . . . 1052 Madison Ave., Memphis, Tenn., ALR
McLAUGHLIN, P. A.,
73 S. Washington St., Wilkes-Barre, Pa., OALR
*McLAURIN, JOHN G. (1926)
1301 Medical Arts Bldg., Dallas, Texas
*McLOONE, JOHN J. (1926) 611 Heard Bldg., Phoenix, Ariz.
*McMAHON, B. J. (1926)
604 Missouri Theatre Bldg., St. Louis, Mo.
McMURRAY, J. B.,
Washington Trust Bldg., Washington, Pa., OALF
*McNICHOLS, WILLIAM A. (1925)
Dixon Nat'l. Bk. Bldg., Dixon, Ill., ALR
McREYNOLDS, G. S. . . . . State Bank Bldg., Temple, Tex., OALR
McREYNOLDS, J. O. . . . . Trust Bldg., Dallas, Tex., OALR
MACFARLAN, D. (1916) 1805 Chestnut St., Philadelphia, Pa., OALR
MACFARLANE, P. B. . . 152 St. James St., S., Hamilton, Can., OALR
MACKENZIE, ALICE V. . . 1724 Spruce St., Philadelphia, Pa., OALR
MACKENZIE, GEORGE W. (1912)
1724 Spruce St., Philadelphia, Pa., OALR
MAC LACHLAN, A. A. . . . . May Bldg., Pittsburgh, Pa., ALR
MACMILLAN, JOHN A. (1925)
129 Stanley St., Montreal, Canada, Op.
MACNIE, J. S. . . . . 74 S. 11th St., Minneapolis, Minn., OALR
MADDEN, REED. . . . . Allen Bldg., Xenia, O., OALR
*MAGGARD, DELANO I., 1127 N. Emporia Ave., Wichita, Kan., OALR
*MAGHY, CHARLES A. (1926)
Med. Office Bldg., Los Angeles, Cal.
MAIDEN, S. D. . . . . 217 Merriam Blk., Council Bluffs, Ia., OALR
MAIRE, L. E. . . . . Kresge Bldg., Detroit, Mich., Op.A
MALONEY, THOS. J. . . . . 1032 Lowry Bldg., St. Paul, Minn., OALR

*MANN, R. H. T.(1913)
State Natl. Bk. Bldg., Texarkana, Tex., OALR
MARCH, CLARA A.....465 Ashland Ave., Buffalo, N. Y., Op.
*MARKEL, J. C.....Westinghouse Bldg., Pittsburgh, Pa., Op.
MARLOW, FRANK W.(1925)
University Bldg., Syracuse, N. Y., Op.
MARQUIS, G. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
MARSHALL, GEORGE G.....Gryphon Bldg., Rutland, Vt., OALR
MARSHALL, W.....Diamond Bank Bldg., Pittsburgh, Pa., ALR
MARTIN, ANCIL.....207 Goodrich Bldg., Phoenix, Ariz., Op.A
MARTIN, H. H.....247 Bull St., Savannah, Ga., OALR
MATHENY, R. C.....306 E. Main St., Galesburg, Ill., OALR
MATHESON, J. P.(1915)
511 Independence Bldg., Charlotte, N. C., OALR
MATTHEWS, J....Metropolitan Bldg., Minneapolis, Minn., LR
MAULDIN, L. O.....Wallace Bldg., Greenville, S. C., OALR
MAUMENEE, A. E.....156 St. Francis St., Mobile, Ala., OALR
MAXEY, E. E.....4 Electric Bldg., Aberdeen, Wash., OALR
MAY, C. H.....698 Madison Ave., New York City, N. Y., Op.
MAY, J. V.....First Nat'l. Bank Bldg., Marinette, Wis., OALR
*MAY, JAMES W.....802 Elks Bldg., Kansas City, Kan., Op.
MAYBAUM, J. L.....12 E. 86th St., New York City, N. Y., ALR
MEANOR, WM. C., 619 Westinghouse Bldg., Pittsburgh, Pa., Op.
MEANS, C. S.,
1006 Pacific-Southwest Bldg., Long Beach, Cal., OALR
MEIERHOF, E. L.,
1140 Madison Ave., New York City, N. Y., OALR
MERCER, RAY.....Stein Bldg., Quincy, Ill., OALR
MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
MERRILL, H. G.....35 East, 2 South St., Provo, Utah, OALR
MERRILL, W. H.(1906)
The Bay State Bldg., Lawrence, Mass., OALR
MESSIER, A. EUGENE(1925)
303 Park Bldg., Worcester, Mass., OALR
MESSINGER, H. C.....170 Broad St., Providence, R. I., Op.
METZ, R. B.....Guardian Bldg., Cleveland, O., Op.
METZENBAUM, M.....Rose Bldg., Cleveland, O., ALR
MIDDLETON, A. B.....201 Sterry Blk., Pontiac, Ill., OALR
MIDDLETON, HARRY E.....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R.....West Palm Beach, Fla., OALR
MILLER, C. E....305 Western Reserve Bldg., Muncie, Ind., OALR
MILLER, C. M.....Stuart Circle Hospital, Richmond, Va., OALR
MILLER, E. B.....2008 Walnut St., Philadelphia, Pa., Op.
MILLER, HUGH....520 Chambers Bldg., Kansas City, Mo., OALR
*MILLETTE, J. W.....58 Cambridge Ave., Dayton, O., OALR
MINER, S. G.....2641 E. Jefferson, Detroit, Mich., ALR
MINOR, C. L.....Fairbanks Bldg., Springfield, O., OALR
MINOR, H. F.....Randolph Bldg., Memphis, Tenn., OALR
*MINSKY, HENRY(1926)
215 W. 88th St., New York City, N. Y.
MINTON, WM. H.....Bartlett Bldg., St. Joseph, Mo., OALR

MITCHELL, E. K....710 W. Lehigh Ave., Philadelphia, Pa., ALR
MITHOEFER, W.....19 Garfield Pl., Cincinnati, O., ALR
MITTENDORF, A. D..399 Park Ave., New York City, N. Y., Op.
MITTENDORF, W. K.,
155 E. 53rd St., New York City, N. Y., Op.A
MOISE, A. B.....Shreveport, La., ALR
MOLT, W. F...Newton Claypool Bldg., Indianapolis, Ind., ALR
*MONAGHAN, D. G.....535 Majestic Bldg., Denver, Colo., Op.
MONAHAN, J. A.....181 Chestnut St., Clinton, Mass., OALR
MONOSMITH, O. B.....424 Broadway, Lorain, O., OALR
MONSON, S. H.....Ainsfield Bldg., Cleveland, O., OALR
MOORE, G. A.....Bank Bldg., Palmer, Mass., OALR
MOORE, GEORGE H.....Schuylkill Bldg., Pottsville, Pa., OALR
MOORE, JOHN D.(1925)
6 Washington St., Bloomfield, N. J., OALR
MOORE, T. K...416 Central Sav. & Trust Bldg., Akron, O., OALR
MOORE, T. W.....1050 Fifth Ave., Huntington, W. Va., OALR
MOORE, W. F.....255 S. 16th St., Philadelphia, Pa., ALR
MORGAN, J. A.....Young Bldg., Honolulu, T. Hawaii, OALR
*MORRIS, A. G.....303 Alexander St., Rochester, N. Y., ALR
MORRISON, F. A.....Willoughby Bldg., Indianapolis, Ind., Op.
*MORRISON, WM. W.(1926).39 E. 50th St., New York City
MORSE, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
MORSMAN, L. W.....203 Congdon Bldg., Hibbing, Minn., OALR
MORTON, HOWARD MCI.,
Metropolitan Bk. Bldg., Minneapolis, Minn., OALR
MORTON, JOHN B.....90 E. Warren Ave., Detroit, Mich., OALR
*MOSHER, H.P.....828 Beacon St., Boston, Mass., ALR
MOSS, HOWARD S.....835 Rialto Bldg., Kansas City, Mo., OALR
MOSS, R. E.....Hicks Bldg., San Antonio, Tex., OALR
MOTT, JOHN S.....214 Argyle Bldg., Kansas City, Mo., OALR
MOULTON, EVERETT C.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
MOULTON, H.,
Merchants Natl. Bank Bldg., Ft. Smith, Ark., OALR
*MULLIN, W. V.(1915).....Cleveland Clinic, Cleveland, O., ALR
MULSOW, JOHN E.....230 Grand Ave., Milwaukee, Wis., OALR
*MUNCASTER, S. B.1026 16th St., N. W., Washington, D. C., Op.
MUNCY, W. M.....23 Waterman St., Providence, R. I., OALR
*MUNDT, G. H.(1912)..25 E. Washington St., Chicago, Ill., OALR
MURDOCH, J. F.....Bessemer Bldg., Pittsburgh, Pa., Op.
MURPHY, J. G.....Murchison Bldg., Wilmington, N. C., OALR
MURPHY, W. E.....7th and Race Sts., Cincinnati, O., OALR
MURRAY, ALFRED N.....4654 Sheridan Rd., Chicago, Ill., OALR
*MURRAY, WM. R.(1900)
1009 Nicollet Ave., Minneapolis, Minn., OALR
MUSSUN, WILLIAM G.(1924) Osborn Bldg., Cleveland, O., ALR
MYERS, ELMER A.,
407 Board of Trade Bldg., Superior, Wis., ALR
*MYERS, E. L.(1926).....207 Wall Bldg., St. Louis, Mo.

*MYERS, JOHN L.(1920)
815 Shukert Bldg., Kansas City, Mo., OALR
MYERSON, M. C.....198 Lincoln Place, Brooklyn, N. Y., ALR

N

NADEAU, E. G....Bellin-Buchanan Bldg., Green Bay, Wis., OALR
NAFTZGER, J. B.....Davidson Bldg., Sioux City, Ia., OALR
NANCE, W. O.....30 N. Michigan Blvd., Chicago, Ill., OALR
*NEEPER, E. R.,
708 Ex. Nat'l. Bank Bldg., Colorado Springs, Colo., Op.
NEHER, E. M(1926)...Boston Bldg., Salt Lake City, Utah
*NEIGHBORS, CLARENCE A.,
Underwood Bldg., Ottawa, Kan., OALR
*NEW, GORDON B.....Mayo Clinic, Rochester, Minn., LR
NELSON, C. F.....Schofield Bldg., Cleveland, O., OALR
NELSON, L. A.....Lowry Bldg., St. Paul, Minn., Op.A
NELSON, LLOYD L.....Norfolk, Neb., OALR
NEWELL, W. S.....1209 Vermont Ave., Washington, D. C.,
*NEWHART, H.(1905) Donaldson Bldg., Minneapolis, Minn., ALR
NEWTON, FRANK H.(1925)
907 Mercantile Bk. Bldg., Dallas, Tex., OALR
NEWTON, L. A.(1919)
709 Medical Arts Bldg., Oklahoma City, Okla., OALR
NISSELSON, MAX, 616 Madison Ave., New York City, N. Y., OALR
NOONAN, WILLIAMS J.(1905)
4654 Sheridan Rd., Chicago, Ill., OALR
*NORCROSS, EDWARD P.(1924)
30 N. Michigan Ave., Chicago, Ill., ALR
*NORTHCOTT, THOMAS A.(1926)
17 E. 38th St., New York City, N. Y.
NORTON, ROY R.....107 B. E. State St., Sharon, Pa., OALR
NOVAK, FRANK, JR....30 N. Michigan Blvd., Chicago, Ill., ALR
NOYES, G. L.....Exchange Bank Bldg., Columbia, Mo., OALR
NUTTER, C. F.....16 Amherst St., Nashua, N. H., Op.

O

*O'BRIEN, C. S.(1924).....Iowa City, Iowa, Op.
O'BRIEN, S. A..400 1st Nat'l. Bank Bldg., Mason City, Ia., OALR
OBERG, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR
O'CONNOR, D. F.....671 Broad St., Newark, N. J., OALR
*O'CONNOR, RODERIC.....1904 Franklin St., Oakland, Cal., Op.
O'DELL, TIMOTHY(1920)
112A Madison Ave., Aurora, Mo., OALR
OERTEL, T. E.....638 Green St., Augusta, Ga., OALR
OESTERLING, H. E.....Winter Park, Fla., OALR
*OFFUTT, W. N.(1905)
406 Security Trust Bldg., Lexington, Ky., OALR

OLSHO, SYDNEY L.....235 S. 15th St., Philadelphia, Pa., OALR
OPPENHEIMER, S...45 E. 60th St., New York City, N. Y., OALR
*ORCUTT, D.C.(1912)...30 N. Michigan Ave., Chicago, Ill., Op.
O'REILLY, C. A.(1911)
520 Med. Arts Bldg., Philadelphia, Pa., ALR
O'REILLY, W. F.(1913)...44 S. Common St., Lynn, Mass., OALR
ORTON, H. B.....671 Broad St., Newark, N. J., ALR
OVERMAN, F. V...Hume-Mansur Bldg., Indianapolis, Ind., ALR
OWEN, F. S.....Medical Arts Bldg., Omaha, Neb., OALR

P

PACKARD, LOUIS(1926).....Sterling, Colo.
PAGE, L.....Hume-Mansur Bldg., Indianapolis, Ind., ALR
*PAINTER, A. M.(1918)
418 Bryant Bldg., Kansas City, Mo., ALR
PALMER, ARTHUR(1926)
2178 Broadway, New York City, N. Y.
PALMER, EDW. M.(1919) 629 Beacon Bldg., Wichita, Kan., OALR
PARK, J. W.(1910).....32 N. Second St., Harrisburg, Pa., OALR
*PARKER, W. R.....David Whitney Bldg., Detroit, Mich., Op.
PARRISH, MAJ. ROBERT E., M. C., U. S. A.,
Walter Reed Gen. Hospital, Washington, D. C., ALR
PARSONS, J. G.....Lewistown, Mont., OALR
PATTERSON, E. J.....Westinghouse Bldg., Pittsburgh, Pa., ALR
*PATTERSON, J. A.(1904)
Burns Bldg., Colorado Springs, Colo., OALR
PATTERSON, JOSEPH M.,
518 Bryant Bldg., Kansas City, Mo., OALR
PATTERSON, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
*PATTON, J. M.(1910)...Medical Arts Bldg., Omaha, Neb., Op.
PAYNE, A. L.(1908)..Opera House Blk., Eau Claire, Wis., OALR
PAYNE, RICHARD J.(1925)
University Club Bldg., St. Louis, Mo., ALR
PEARLMAN, SAMUEL J.....29 E. Madison St., Chicago, Ill., ALR
*PEARSON, W. W.(1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N.(1920).....W. 7th St., Charlotte, N. C., OALR
PEIRCE, HOWARD W., David Whitney Bldg., Detroit, Mich., ALR
*PEMBER, AUBREY H.(1926)
500 W. Milwaukee St., Janesville, Wis.
*PENDEXTER, RALPH S.(1926)
1424 K St., N. W., Washington, D. C.
PENTECOST, R. S.(1926)...90 College St., Toronto, Canada,
*PETER, LUTHER C.(1914)
N. E. cor. Chestnut & 20th Sts., Philadelphia, Pa., Op.
PETERMAN, H. E.(1912)
518 No. Charles St., Baltimore, Md., OALR
PETERSON, R. A.....University Hosp., Nankin, China

PFAFFLIN, C. A.,
Newton Claypool Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....Francis Bldg., Louisville, Ky., Op
PFINGSTEN, C. F.....508 N. Grand Ave., St. Louis, Mo., ALR
PHELPS, KENNETH A. (1920)
730 La Salle Bldg., Minneapolis, Minn., OALR
PHILLIPS, W. C.....40 W. 47th St., New York City, ALR
PHILLIPS, W. L.....759 Richmond Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PITKIN, C. E.....Osborn Bldg., Cleveland, O., ALR
*POLLOCK, H. L. (1911)....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., OALR
PORTER, E. H. (1912).....85 Madison St., Tiffin, O., OALR
*PORTER, JESSE B. (1920).....415 S. 2nd St., Elkhart, Ind., OALR
PORTER, L. B. (1913)....195 Thayer St., Providence, R. I., OALR
POSEY, WM. CAMPBELL,
21st and Chestnut Sts., Philadelphia, Pa., Op.
POST, LAWRENCE (1920)
520 Metropolitan Bldg., St. Louis, Mo., Op.
POST, MARTIN H. (1918)
520 Metropolitan Bldg., St. Louis, Mo., Op.
*POST, WINFRED B. (1921)
First National Bank Bldg., Carthage, Mo., OALR
POTTER, GEORGE B. (1921)
402 City Natl. Bank Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER (1918)
Granate and Alaska Sts., Butte, Mont., OALR
POTTS, J. B.....Medical Arts Bldg., Omaha, Neb., ALR
POWERS, E. (1916)
Central Nat'l Bank Bldg., Carthage, Mo., OALR
PRANGEN, AVERY DE HART..Mayo Clinic, Rochester, Minn., Op.
PRATT, F. J., JR. (1912)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
PRATT, J. A. (1910)
Metropolitan Bank Bldg., Minneapolis, Minn., OALR
PRENDERGAST, D.....850 Hanna Bldg., Cleveland, O., OALR
PRICE, N. W. (1908).....445 3rd St., Niagara Falls, N. Y., OALR
PROETZ, ARTHUR W.,
University Club Bldg., St. Louis, Mo., ALR
PROUD, WILLARD C. (1920)
Phys. & Surg. Bldg., St. Joseph, Mo., OALR
PROUT, A. W.....370 E. Town St., Columbus, O., OALR
PYLE, W. (1915). 18th and Exchange Pl., Jersey City, N. J., OALR

Q

QUINBY, WM. O'G. (1919)...14 James St., Newark, N. J., OALR
QUITTNER, S. S.....3912 Prospect Ave., Cleveland, O., OALR

R

RADCLIFFE, MCCLUNEY,
1906 Chestnut St., Philadelphia, Pa., Op.
*RALLS, C. T.(1916)..1st Nat'l. Bank Bldg., Winfield, Kas., OALR
*RALSTON, WALLACE W.(1918)
1300 Walker Ave., Houston, Tex., Op.
RAVDIN, B. D.....Citizens Natl. Bk. Bldg.,Evansville, Ind., OALR
RAVDIN, M.....Citizens Natl. Bk. Bldg., Evansville, Ind., OALR
RAY, V.(1901).....19 W. 7th St., Cincinnati, O., OALR
REAVES, CHAS. R.....Box 187, Knoxville, Tenn., OALR
REAVES, W. P.....Reaves Infirmary, Greensboro, N. C., OALR
REBUCK, C. S.....412 N. Third St., Harrisburg, Pa., OALR
RECTOR, A. E.....112 College Ave., Appleton, Wis., OALR
REEDER, J. E.(1916).....Davidson Bldg., Sioux City, Ia., OALR
*REESE, M. D.(1926)..3527 Hamilton St., Philadelphia, Pa.
REESE, R. G.....50 W. 52nd St., New York City, N. Y., Op.
REESE, WARREN S.(1921) 230 S. 21st St., Philadelphia, Pa., Op.
*REGER, H. S.(1908).641 Lakeview Ave., Jamestown, N. Y., OALR
REICHE, O. C.....328 West Broad St., Hazleton, Pa., OALR
REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
REINEKE, G. F.(1926).....New Ulm, Minn.
RENAUD, G. L.....Fine Arts Bldg., Detroit, Mich., OALR
RENNER, W. S.(1900)..341 Linwood Ave., Buffalo, N. Y., ALR
REPASS, ROBERT E.,
1003 Hume-Mansur Bldg., Indianapolis, Ind., ALR
*REULING, F. HAROLD(1924) 721 Black Bldg., Waterloo, Ia., OALR
REYNOLDS, H. G.....City Nat'l. Bank Bldg., Paducah, Ky., Op.A
*REYNOLDS, J. S.(1918)
816 La Salle Bldg., Minneapolis, Minn., OALR
RICE, C. P.....7 Sterns Bldg., Wahpeton, N. D., OALR
RICE, G. B.(1916) 270 Commonwealth Ave., Boston, Mass., ALR
RICE, JOHN E.....390 Main St., Worcester, Mass., OALR
RICE, R. H.....Coshwell Bldg., Milwaukee, Wis., Op.AL
RICHARDS, LYMAN, G.(1924)
270 Commonwealth Ave., Boston, Mass., ALR
RICHARDSON, C. W.,
1317 Connecticut Ave., Washington, D. C., ALR
RICHARDSON, J. J.,
1509 Ave. of Presidents, Washington, D. C., ALR
RICHARDSON, SHALER A.(1925)
111 W. Adams St., Jacksonville, Fla., Op.
*RICHIE, L. T.(1916)...McCormick Bldg., Trinidad, Colo., OALR
RIDEOUT, W. J.....90 Stephenson St., Freeport, Ill., OALR
RIDPATH, R. F.....1928 Chestnut St., Philadelphia, Pa., ALR
RIEGER, WALTER H.....Hanna Bldg., Cleveland, O., ALR
RINDLAUB, ELIZABETH P.,
DeLendrecie Block, Fargo, N. D., OALR
RINDLAUB, JOHN H.....DeLendrecie Block, Fargo, N. D., OALR
RINDLAUB, MARTIN P., JR.,
DeLendrecie Block, Fargo, N. D., OALR

RINGLE, C. A.....Coronado Bldg., Greeley, Colo., ALR
RINGUEBERG, E. N. S.....13 Main St., Lockport, N. Y., Op.
RISDON, ERNEST F.(1924) 30 Avenue Rd., Toronto, Canada, ALR
DE RIVER, JOSEPH PAUL,
U. S. Veterans' Bureau, San Francisco, Cal., OALR
*ROBERTS, S. E.(1916)
1810 Federal Reserve Bank Bldg., Kansas City, Mo., ALR
ROBERTS, W. H.....461 E. Colorado St., Pasadena, Cal., OALR
ROBERTSON, C. M.....30 N. Michigan Ave., Chicago, Ill., OALR
*ROBERTSON, E. N.....132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
ROBINSON, H. T.....Medical Bldg., Cumberland, Md., OALR
*ROBINSON, ROBERT E.(1920)
205½ E. Bremer Ave., Waverly, Ia., OALR
ROGERS, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
ROGERS, J. T.....758 Sherbrooke St., W., Montreal, Can., ALR
ROLLER, L. A.....122 Monroe St., Grand Rapids, Mich., OALR
ROMMEL, JOHN C....4501 N. Broad St., Philadelphia, Pa., OALR
ROOKER, A. L.(1920)
225 Gluck Bldg., Niagara Falls, N. Y., ALR
ROOST, F. H.(1914).....515 Trimble Blk., Sioux City, Ia., OALR
ROSENBAUM, J.....206 Bishop St., Montreal, Can., Op.
ROTH, ALBERT W.(1920)...303 Palace Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J..239 Lowry Bldg., St. Paul, Minn., OALR
*ROTT, O. M.(1915).....Paulsen Bldg., Sponkane, Wash., OALR
ROW, G. S.(1911).Hume-Mansur Bldg., Indianapolis, Ind., Op.
ROWLAND, J. F.....Thompson Bldg., Hot Springs, Ark., OALR
ROWLAND, WILLIAM D..220 Clarendon St., Boston, Mass., Op.
RUBENDALL, C.(1915)
Medical Arts Bldg., Omaha, Neb., OALR
RUBY, F. M.....Baru Bldg., Long View, Wash., OALR
RUCKER, E. W., JR.,
926 Woodward Bldg., Birmingham, Ala., ALR
RUSH, CALVIN C.....342 Main St., Johnstown, Pa., ALR
RUSSELL, EDWIN L.(1920)
402 Argyle Bldg., Kansas City, Mo., OALR
RUSSELL, J. A.....206 Masonic Temple, Erie, Pa., ALR
RUSSELL, RALPH E.....Waterloo, Iowa, OALR
RUST, E. G.....Hanna Bldg., Cleveland, O., Op.A
RUTHERFORD, CYRUS W.(1925)
Pennway Bldg., Indianapolis, Ind., Op.
RYDER, D. R.....151 Rock St., Fall River, Mass., ALR
RYERSON, FRANK L.(1925)
307 Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY G.(1920)
440 Clinton Ave., West Hoboken, N. Y., OALR
*SALINGER, SAMUEL(1920)
25 E. Washington St., Chicago, Ill., ALR

SALLANDER, FREDERICK W.(1919)
303 Security Bank Bldg., Sioux City, Ia., OALR
SANDERSON, D. D.(1917).....Funke Bldg., Lincoln, Neb., OALR
SANDERSON, H. H...David Whitney Bldg., Detroit, Mich., OALR
SARTAIN, PAUL J.(1921)
2006 Walnut St., Philadelphia, Pa., Op.
SATTERLEE, R. H.....187 Delaware Ave., Buffalo, N. Y., Op.
SAUER, W. E.....Humboldt Bldg., St. Louis, Mo., ALR
*SAUER, W. W.(1910).....231 Third Ave., Marietta, O., OALR
SAVERY, C. E.....126 N. Lafayette St., South Bend, Ind., OALR
*SCAL, JOSEPH C.(1926)
44 W. 74th St., New York City, N. Y.
*SCALES, H. L.(1920)
504-5 Rorabaugh-Wiley Bldg., Hutchinson, Kas., OALR
*SCALES, J. L..Commercial Natl. Bk. Bldg., Shreveport, La., OALR
*SCALES, J. W.(1896)....202½ Main St., Pine Bluff, Ark., OALR
SCARLET, HUNTER W.(1919)
230 S. 21st St., Philadelphia, Pa., Op.
SCHAPPERT, N. L.59 S. Washington St., Wilkes-Barre, Pa., OALR
SCHENCK, CHARLES P.(1920)
301 Texas St. Bank Bldg., Fort Worth, Tex., OALR
SCHILD, E. H.(1906)
Cleveland Ave. and 9th St., Canton, O., Op.A
SCHILLER, A. NOAH,
255 West End Ave., New York City, N. Y., ALR
SCHLANSER, ADAM E.(1921).....Ft. Leavenworth, Kas., OALR
SCHLEGEL, H. T.....501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. W.....138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN(1925)
1000 Park Ave., New York City, N. Y., Op.
SCHNEIDEMAN, T.B., Professional Bldg., Philadelphia, Pa., Op.
SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
*SCHRANT, J. H.,
Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
SCHULZE, E. C.(1926).....Fox Bldg., McKinney, Tex.
SCHURTER, M. A.....2762 E. 2nd St., Long Beach, Cal., OALR
SCHUSTER, STEPHEN A..1st Natl. Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H.....712 Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, FREDERICK A.(1919)
601 Metropolitan Bldg., St. Louis, Mo., Op.
SCHWEINITZ, GEO. E. DE,
1705 Walnut St., Philadelphia, Pa., Op.
SCHWENK, P. N. K..1417 N. Broad St., Philadelphia, Pa., Op.
SCOTT, JOHN R.(1920).....201 S. Main St., Ottawa, Kan., OALR
SCOTT, M. E.(1926).....1st Natl. Bk. Bldg., Massillon, O.
SCROGGS, J. J.....218 E. Third St., Beaver, Pa., OALR
SEAMAN, GILBERT E.,
309 Goldsmith Bldg., Milwaukee, Wis., OALR
SEARCY, H. B.....807 Greensboro Ave., Tuscaloosa, Ala., OALR
*SEARS, WM. H.(1910).....514 Penn St., Huntingdon, Pa., OALR
SEAY, D. E.....Dallas Co. State Bk. Bldg., Dallas, Tex., OALR

SECOY, FRANK L. (1922) .525 Francis Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W. (1917) ..... Box 494, Ionia, Mich., OALR
SEDWICK, W. A. ..... Imperial Bldg., Denver, Colo., OALR
*SEEGMAN, S. ..... Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
SEELYE, WALTER K. (1920)
806 American Bank Bldg., Seattle, Wash., OALR
SELFRIDGE, G. L. ..... Schroth Bldg., San Francisco, Cal., ALR
*SELIGSTEIN, MILTON B. (1926)
801 Union Planters Bk. Bldg., Memphis, Tenn.
SELLERS, LYLE M. ..... Medical Arts Bldg., Dallas, Texas, ALR
SENER, WALTER J. ..... 130 S. 18th St., Philadelphia, Pa., OALR
SERNOFFSKY, ISAAC ..... 37 Allen St., Buffalo, N. Y., Op.
SEWALL, E. C. ..... Lane Hospital, San Francisco, Cal., OALR
SEYDELL, E. M. (1916)
1021 First Natl. Bk. Bldg., Wichita, Kas., ALR
SHACKLETON, W. E. (1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J. (1913) ..... Stark Bldg., Louisville, Ky., OALR
SHAHAN, W. E. ..... Metropolitan Bldg., St. Louis, Mo., Op.
SHANAHAN, T. J. (1914) .. 419 Boylston St., Boston, Mass., ALR
SHANKLIN, E. M. ..... 575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. ..... 104 Copley Rd., Upper Darby, Pa., Op.
SHAPERO, I. M. (1919) 289 Alexander St., Rochester, N. Y., OALR
SHARP. B. S. (1926) .... 218½ E. College St., Iowa City, Ia.
SHARRETT, GEORGE O. (1921)
119 Bedford St., Cumberland, Md., OALR
SHASTID, T. H. (1913) ... 400 Lyceum Bldg., Duluth, Minn. Op.
SHAWHAN, GLENN E. ..... Nampa, Ida., OALR
*SHEA, J. J. (1916) .... 1018 Madison Ave., Memphis, Tenn., OALR
SHEAHAN, WM. L. (1926)
59 College St., New Haven, Conn.
SHELLMAN, J. L. (1917) .... Lowry Bldg., St. Paul, Minn., OALR
SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
*SHERMAN, MAJ. A. EDWARD, M.C., U. S. A. (1913)
Ft. Leavenworth, Kansas, OALR
SHERMAN, E. S. (1912) ..... 671 Broad St., Newark, N. J., OALR
*SHIELDS, J. M. ..... Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE ..... Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H. .... 629 Guardian Bldg., Cleveland, O., Op.
SHIREY, OSCAR M. (1925) ..... Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Veterans' Hospital No. 67,
11th and Harrison Sts., Kansas City, Mo., OALR
SHOEMAKER, W. A. ..... Carleton Bldg., St. Louis, Mo., Op.
SHORT, Z. N. ..... Dugan Stuart Bldg., Hot Springs, Ark., OALR
SHREVE, O. M. (1905) ..... 162 W. 8th St., Erie, Pa., OALR
SHULES, EDWIN J. G. (1920)
502 N. Washington, Wellington, Kansas, OALR
SHUMAN, G. H. (1917) .. 1105 Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, DAVID L. .. 517 Shukert Bldg., Kansas City, Mo., OALR
*SHURLY, B. R. ..... Shurly Bldg., Detroit, Mich., ALR
SHUTE, D. K. ..... 1717 DeSales St., Washington, D. C., Op.

*Siegel, F. X.(1922)
2600 Union Central Bldg., Cincinnati, O., OALR
Silbernagel, C. E.....283 E. State St., Columbus, O., OALR
Simonds, O. F.....Hanna Bldg., Cleveland, O., Op.
Simpson, H. Lee,
1413 David Whitney Bldg., Detroit, Mich., ALR
*Simpson, W. L.(1911) Columbian Tower, Memphis, Tenn., ALR
Singleton, E. M.(1897)
111 E. Church St., Marshalltown, Ia., OALR
*Skillern, R. H.(1910).1811 Spruce St., Philadelphia, Pa., LR
Skillern, Samuel R., Jr.(1925)
2017 Walnut St., Philadelphia, Pa., ALR
*Sleight, R. D.(1906).....Post Bldg., Battle Creek, Mich., Op.
Sloan, Henry L.(1919)
Independence Bldg., Charlotte, N. C., Op.
Slocum, G.....328 E. Huron St., Ann Arbor, Mich., Op.
Sluder, G.....3542 Washington Ave., St. Louis, Mo., ALR
Smith, Arthur E.,
1016 Donaldson Bldg., Minneapolis, Minn., OALR
Smith, D.....834 Myrtle Ave., Bridgeport, Conn., Op.A
*Smith, D. S.(1917)....State Bank Bldg., La Crosse, Wis., OALR
Smith, E. T.....Connecticut Mut. Bldg., Hartford, Conn., Op.
Smith, G. B.(1914)...407 1st Natl. Bk. Bldg., Rome, Ga., OALR
*Smith, H. A.....Colo. Bank Bldg., Delta, Colo., OALR
Smith, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
Smith, R. C.....Board of Trade Bldg., Superior, Wis., OALR
Smith, S. McC.....1429 Spruce St., Philadelphia, Pa., ALR
Smith, S. S.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
Smith, V. C.(1912) 722 Audubon Bldg., New Orleans, La., Op.
Sneed, Carl M.....506 W. Broadway, Columbia, Mo., OALR
*Snell, A. C.(1925)..53 S. Fitzhugh St., Rochester, N. Y., Op.
Snyder, W. H.....212 Ontario St., Toledo, O., OALR
Somberg, Joseph S.....60 E. 67th St., New York, N. Y., Op.
*Sonnenschein, R.(1915).29 E. Madison St., Chicago, Ill., ALR
Spake, L. B.(1920)
522 Minnesota Ave., Kansas City, Kans., ALR
Spalding, J. A.(1896)....627 Congress St., Portland, Me., Op.A
*Spencer, F. R.(1904)...Physicians Bldg., Boulder, Colo., OALR
Spencer, W....1830 S. Rittenhouse Sq., Philadelphia, Pa., OALR
Spengler, J. A.....423 S. Main St., Geneva, N. Y., OALR
Spitze, E. C.(1916)....Murphy Bldg., East St. Louis, Ill., Op.
Stahlman, F. C.....318 Fifth St., Charleroi, Pa., OALR
Stanberry, H.....Doctors Bldg., Cincinnati, O., Op.AR
Stanford, J. B.....Exchange Bldg., Memphis, Tenn., OALR
Stark, Jesse B.(1925)..270 Park Ave., New York, N. Y., Op.
*Stauffer, F..801 Deseret Bk. Bldg., Salt Lake City, Utah, OALR
Stegman, L. V.....Sanitarium, Battle Creek, Mich., Op.
Steim, J. M..First Natl. Bk. Bldg., New Kensington, Pa., Op.
Stein, Jules C.(1924) 58 E. Washington St., Chicago, Ill., Op.
Stein, O. J.....30 N. Michigan Blvd., Chicago, Ill., ALR
Steinfeld, A. L.(1922).....125 15th St., Toledo, O., OALR

STELLE, HARRY L....207 Commerce Bldg., Pittsburg, Kan., OALR
STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
STEVENSON, W. D.....Majestic Bldg., Quincy, Ill., OALR
STEWART, WALTER E....Tribune Bldg., Terre Haute, Ind., OALR
STIEREN, E.(1905) 626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILES, C. M.....1831 Chestnut St., Philadelphia, Pa., Op.
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
STIMSON, GEORGE W.(1919) Packard Apts., Warren, Ohio, ALR
STOCKLER, J. A.....332 North Fifth St., Reading, Pa., OALR
STOKES, WM. H.(1926)....4105 Live Oak St., Dallas, Tex.
STOTTER, ARTHUR L.....1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S....Medical Arts Bldg., Philadelphia, Pa., ALR
*STRADER, G. L.(1904)..408 Hynds Bldg., Cheyenne, Wyo., OALR
STRAUSS, JEROME F.(1925)
104 S. Michigan Blvd., Chicago, Ill., ALR
STRICKER, L.....The Groton, Cincinnati, O., Op.
*STRICKLER, D. A.....Republic Bldg., Denver, Colo., Op.
*STRONG, J. C.(1915)...810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD F.(1926)
805 Med. Arts Bldg., Dallas, Tex.
STROUSE, FRED. M.....1301 Spruce St., Philadelphia, Pa., ALR
STROUT, E. S.(1918)...74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. ELMER...Donaldson Bldg., Minneapolis, Minn., OALR
STUCKY, J. A.(1896)
311 Guaranty Bank Bldg., Lexington, Ky., OALR
STUCKY, W. S...311 Guaranty Bank Bldg., Lexington, Ky., OALR
STUEBER, F. G.(1905).....209 W. North St., Lima, O., OALR
STUEBER, P. J.(1917).....209 W. North St., Lima, O., OALR
STURGIS, LEIGH F.(1920)
128 W. 87th St., New York City, N. Y., ALR
STURM, S. A.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, G. F.(1896)...25 E. Washington St., Chicago, Ill., Op.
SULLIVAN, JOHN J., JR..Traders Bank Bldg., Scranton, Pa., ALR
SULZER, G. A.....Medical Arts Bldg., Columbus, O., OALR
SULZMAN, F. M.(1922).....1831 Fifth Ave., Troy, N. Y., OALR
SWAN, C. J.(1911).....The Gables Bldg., Evanston, Ill., OALR
*SWEET, R. B.(1926)
1006 Pacific S. W. Bk. Bldg., Long Beach, Calif.
SWEET, W. M.....1205 Spruce St., Philadelphia, Pa., Op.
SWETNAM, C. R. K.(1915) Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F.(1919)
607 Union Central Bldg., Cincinnati, O., ALR
*TAQUINIO, GEORGE J. (1920)
202 Audubon Bldg., New Orleans, La., ALR
TARUN, W.....605 Park Ave., Baltimore, Md., Op.A
TAYLOR, CHAS. B.(1921).....Ennis Bldg., Ottumwa, Ia., ALR
TAYLOR, L. E.(1905)
Marshall Field Annex Bldg., Chicago, Ill., ALR

*Taylor, Lewis H...83 S. Franklin St., Wilkes-Barre, Pa., OALR
Taylor, M. C.....1206 May Bldg., Pittsburgh, Pa., Op.
Thigpen, C. A.....401 So. Court St., Montgomery, Ala., OALR
Thomas, Chas. D...Central Natl. Bank Bldg., Peoria, Ill., OALR
Thomas, Frances W.(1916)..74 S. 5th St., Columbus, O., OALR
Thomas, H. G.....Dalziel Bldg., Oakland, Cal., OALR
Thomason, H. E.....Rialto Bldg., Kansas City, Mo., OALR
Thomasson, W. J.....942 York St., Newport, Ky., OALR
Thompson, E. H.,
Vindonissa Bldg., 19 W. 7th St., Cincinnati, O., OALR
Thompson, G. H.(1909)
18 Ashland St., North Adams, Mass., OALR
Thompson, H. M.(1912)
First Natl. Bank Bldg., Pueblo, Colo., Op.
Thompson, J. J.....3 Park Ave., Mt. Vernon, N. Y., OALR
Thompson, P. H.,
308 Commonwealth Ave., Boston, Mass., Op.
*Tilderquist, D. L.....425 Bradley Bldg., Duluth, Minn., OALR
Timberman, A.....112 E. Broad St., Columbus, O., OALR
Tindolph, L. W.....1st Natl. Bank Bldg., Olean, N. Y., OALR
Tingley, Louisa Paine(1913)
9 Massachusetts Ave., Boston, Mass., Op.
Titsworth, Guy.....111 W. 4th St., Sedalia, Mo., Op.
Tivnen, R. J.....104 S. Michigan Blvd., Chicago, Ill., OALR
*Tobey, G. L.,Jr...270 Commonwealth Ave., Boston, Mass., ALR
Todd, G. C.(1922)
307 Westinghouse Bldg., Pittsburgh, Pa., OALR
Tomassene, Raymond A.,
Wheeling Bank & Trust Bldg., Wheeling, W. Va., OALR
Tomlin, W. S....Hume-Mansur Bldg., Indianapolis, Ind., ALR
Toms, S. W. S(1909).....Nyack-on-Hudson, N. Y., ALR
Tooker, C. W. (1907).....Carleton Bldg., St. Louis, Mo., Op.
Townsend, John F.(1923)
85 Wentworth, Cor. St. Phillips St., Charleston, S. C.,OALR
*Trible, George B.(1920)
2705 Woodley Rd., Washington, D. C., ALR
*Trimble, Clarence S.(1920)
City Natl. Bank Bldg., Emporia, Kan., OALR
Tripp, Ira A.(1920).....436 Rose Bldg., Cleveland, O., OALR
Tripp, Leroy R.....307 Security Bldg., Sioux City, Iowa, OALR
*Tucker, Gabriel F.(1923) 107 Lodges Lane, Cynwyd, Pa., ALR
*Tucker, James C.(1920).....114 So. 6th, Beatrice, Neb., OALR
Tuckerman, W. C.(1911)...Osborn Bldg., Cleveland, O., Op.
Tuckerman, W. H.....Osborn Bldg., Cleveland, O., OALR
Tureman, Herbert G.,
1100 Rialto Bldg., Kansas City, Mo., OALR
Turnbull, F. M.(1916)
504 Providence Bldg., Duluth, Minn., ALR
*Turner, H. H.(1914)
902 Professional Bldg., Pittsburgh, Pa., Op.
Turner, O. W.....Augusta Trust Bldg., Augusta, Me., OALR

U

UPHAM, H. F.....305 Third Ave., Asbury Park, N. J., OALR
UREN, CLAUDE THOMAS
537 City Natl. Bank Bldg., Omaha, Neb., OALR
URNER, M. H....Union Central Life Bldg., Cincinnati, O., OALR

V

VAIL, D. T.....24 E. 8th St., Cincinnati, O., Op.
VAIL, H. H.(1924).....24 E. 8th St., Cincinnati, O., ALR
*VAN KIRK, FRANK J.,
312 Bell. Natl. Bank Bldg., Bellingham, Wash., OALR
VANKIRK, V. E.....Union Arcade Bldg., Pittsburgh, Pa., Op.
VAN POOLE, GIDEON McD.,
Young Bldg., Honolulu, T. Hawaii, OALR
VAUGHAN, G. E.....711 S. 3rd St., Louisville, Ky., OALR
VAUGHN, EDGAR H...113½ W. Ferguson St., Tyler, Tex., OALR
VEASEY, C. A.....Paulsen Bldg., Spokane, Wash., OALR
VINSONHALER, F.....Urquhart Bldg., Little Rock, Ark., OALR
VOIGT, C. B.....1702 Broadway, Mattoon, Ill., OALR
VON COLDITZ, G. F....25 E. Washington St., Chicago, Ill., OALR
*VON DER HEYDT, ROBERT(1926)
25 E. Washington St., Chicago, Ill.

W

*WAGENER, HENRY P.(1926) Mayo Clinic, Rochester, Minn.
WAGERS, ARTHUR J.,
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
WAGNER, C. B.....31 North State St., Chicago, Ill., OALR
WALKER, CHAS. C..Woodward at Warren, Detroit, Mich., Op.
WALKER, C. E.....Jacobson Bldg., Denver, Colo., Op.
WALKER, H. S.....77 E. Washington St., Chicago, Ill., OALR
WALSH, JOSEPH M.....Rapid City, S. D., OALR
WALTER, WILL.....1507 Hinman Ave., Evanston, Ill., OALR
*WATKINS, E. D.(1926)
1412 Exchange Bldg., Memphis, Tenn.,
WATSON, H. D.(1915) 151 Front St., Binghampton, N. Y., OALR
WATSON, R. SEYMOUR,
120 N. Michigan Ave., Saginaw, Mich., OALR
WAUGH, J. M.,
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., LR
*WEAVER, T. W.(1914)....201 N. Main St., Wichita, Kan., Op.
*WEED, H. M.(1908)....196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN H....20 E. 53rd St., New York City, N. Y., Op.
WEEKS, W. W.....20 E. 53rd St., New York City, N. Y., Op.
*WEIH, ELMER PAUL(1920)
602 Argyle Court, Clinton, Iowa, OALR
WEIL, A. L.....717 Audubon Bldg., New Orleans, La., ALR
WEIMER, E. S.....332 Lehigh Ave., Pittsburgh, Pa., Op.
WEISSER, E. A.....Empire Bldg., Pittsburgh, Pa., Op.

*Wetherbee, Charles (1926) ..... Twin Falls, Ida.
Welch, R. S. G. ..... 46 State St. Circle, Annapolis, Md., OALR
Wells, D. W. ..... Hotel Westminster, Boston, Mass., Op.
Wells, G. S. ..... San Marcos Bldg., Santa Barbara, Cal., ALR
Welsh, D. E. (1896)
Powers Theater Bldg., Grand Rapids, Mich., OALR
Welty, C. F. ..... Shreve Bldg., San Francisco, Cal., ALR
Wendel, Jacob S. .... David Whitney Bldg., Detroit, Mich., ALR
Westlake, S. V. ..... Humboldt Bldg., St. Louis, Mo., ALR
Wheeler, Ernest Carlton,
Tacoma Clinic, Tacoma, Wash., OALR
*Wheeler, John M. (1920)
30 W. 59th St., New York City, N. Y., Op.
Wheeler, Merritt W. ..... Lowry Bldg., St. Paul, Minn., OALR
Whelan, George L. (1925)
257 S. 16th St., Philadelphia, Pa., ALR
*Wherry, W. P. ... 1500 Medical Arts Bldg., Omaha, Neb., OALR
Whisnant, A. M. ..... Realty Bldg., Charlotte, N. C., OALR
Whitaker, J. ..... Alleson Hosp., Miami Beach, Fla., OALR
White, C. P. ..... 507 Ford Bldg., Wilmington, Del., OALR
White, E. H. ..... 756 Sherbrook St., Montreal, Can., ALR
White, L. E. (1912)
390 Commonwealth Ave., Boston, Mass., ALR
Wiener, A. (1909) .. 550 Park Ave., New York City, N. Y., OALR
Wiener, M. ..... Carleton Bldg., St. Louis, Mo., Op.
Wilbur, E. P.,
Kalamazoo Natl. Bank Bldg., Kalamazoo, Mich., OALR
Wilder, W. H. ..... 122 S. Michigan Blvd., Chicago, Ill., Op.
Willard, L. M. ..... 520 3rd St., Wausau, Wis., OALR
Williams, Horace J. (1922)
5908 Green St., Germantown, Philadelphia, Pa., ALR
Williams, Thos. J. ... 30 N. Michigan Blvd., Chicago, Ill., OALR
Williams, W. C. ..... Jefferson Bldg., Peoria, Ill., OALR
*Williams, Walter H. (1921)
Castell Bldg., Middletown, O., OALR
*Winter, G. E. (1921) ..... 44 Sun Bldg., Jackson, Mich., OALR
*Winter, John Arthur (1923)
Fidelity Bldg., Duluth, Minn., OALR
Wise, Ralph C. ..... News Bldg., Mansfield, O., OALR
Wold, Karl C. (1924)
7th and Wacouta Sts., St. Paul, Minn., OALR
*Wolfe, C. T. (1916) .... 654 Francis Bldg., Louisville, Ky., OALR
*Wolfe, Otis R. (1919)
908 W. Boone St., Marshalltown, Iowa, OALR
Wood, C. A. ..... 7 W. Madison St., Chicago, Ill., Op.
Wood, D. ..... Donaldson Bldg., Minneapolis, Minn., OALR
Wood, Hilliard ..... Independent Bldg., Nashville, Tenn., OALR
Woodruff, F. E. ..... Metropolitan Bldg., St. Louis, Mo., Op.
*Woodruff, H. W. ..... 305 Heggie Bldg., Joliet, Ill., OALR
Woodruff, T. A. ..... Plant Bldg., New London, Conn., Op.

WOODS, HIRAM.....842 Park Ave., Baltimore, Md., Op.A
WOODS, R. H.....Neustadt Bldg., La Salle, Ill., OALR
*WOODSON, J. M.(1914)
Cor. Avenue G and Fifth, Temple, Tex., OALR
WORMLEY, H. R.....410 Brown Bldg., Rockford, Ill., OALR
WRIGHT, CHAS. D.(1921)
1111 Metropolitan Bank Bldg., Minneapolis, Minn., Op.A
WRIGHT, J. B...Citizens Natl. Bank Bldg., Raleigh, N. C., OALR
WRIGHT, J. W...Central Natl. Bank Bldg., Columbus, O., Op.
WRIGHT, J. W. M.,
Hume-Mansur Bldg., Indianapolis, Ind., ALR
*WRIGHT, W. W.(1926)..143 College St., Toronto, Canada
WÜRDEMANN, H. V.....Cobb Bldg., Seattle, Wash., OALR
WYLIE, C. B.....293 High St., Morgantown, W. Va., OALR

Y

YANKAUER, S...616 Madison Ave., New York City, N. Y., ALR
YAZEL, HERMAN E.,
510-511 Grand Ave. Temple, Kansas City, Mo., OALR
*YOUNG, CHARLES A.(1926)
Gill Mem. E., E. and Throat Hospital, Roanoke, Va.
*YOUNG, H. B.(1897)..204 Jefferson St., Burlington, Iowa, OALR

Z

ZEHNDER, A. CHARLES(1919)
180 Fairmount Ave., Newark, N. J., Op.
ZENTMAYER, WILLIAM..1506 Spruce St., Philadelphia, Pa., Op.
ZINKHAN, ARTHUR M.,
1025 Fifteenth St., N. W., Washington, D. C., ALR

GEOGRAPHIC LIST

ALABAMA

BIRMINGHAM

HARRISON, W. G. ..... Empire Bldg., OALR
RUCKER, EDMOND W. JR. ..... Woodward Bldg., ALR

MOBILE

MAUMENEE, A. E. ..... 156 St. Francis St., OALR

MONTGOMERY

THIGPEN, C. A. ..... 401 S. Court St., OALR

SELMA

KIRKPATRICK, SAMUEL ..... Lauderdale Ave., OALR

TUSCALOOSA

SEARCY, H. B. ..... 807 Greensboro Ave., OALR

ARIZONA

PHOENIX

McLOONE, J. J. ..... Heard Bldg.
MARTIN, ANCIL ..... 207 Goodrich Bldg., Op.A

PRESCOTT

SWETNAM, C. R. K. ..... Masonic Temple, ALR

ARKANSAS

FORT SMITH

BUCKLEY, J. H. ..... 1st National Bank Bldg., OALR
DORENTO, DRED R. ..... Merchants National Bank Bldg., OALR
MOULTON, EVERETT C. ..... Merchants National Bank Bldg., OALR
MOULTON, HERBERT. ..... Merchants National Bank Bldg., OALR

HOT SPRINGS

KING, O. H. ..... Dugan-Stuart Bldg., OALR
ROWLAND, J. F. ..... Thompson Bldg., OALR
SHORT, Z. N. ..... Dugan-Stuart Bldg., OALR

LITTLE ROCK

CALDWELL, ROBERT ..... State National Bank Bldg., OALR
VINSONHALER, FRANK ..... Urquhart Bldg., OALR

PINE BLUFF

SCALES, J. W. ..... 202½ Main St., OALR

ROGERS

KOOBS, H. J. G.....4th and Walnut Sts., OALR

CALIFORNIA

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LAFORCE, BERT D.....1140 Beverly Rd., OALR

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THOMASSON, W. J.....942 York St., OALR

PADUCAH

REYNOLDS, H. G.....City National Bank Bldg., Op.A

LOUISIANA

NEW ORLEANS

BAHN, CHAS. A.....Audubon Bldg., Op.
BLUM, H. N.....Maison Blanche Bldg., Op.
HASPEL, D. M.....1214 Maison Blanche Bldg., ALR
LANDFRIED, C. J....607 Land Title and Guarantee Bldg., ALR
LYNCH, R. C.....Maison Blanche Bldg., ALR
SMITH, V. C.....Audubon Bldg., Op.
TAQUINIO, G. J.....Audubon Bldg., Op.
WEIL, A. L.....717 Audubon Bldg., ALR

SHREVEPORT

MOISE, A. B.....Commercial Natl. Bk. Bldg., ALR
SCALES, J. L.....Commercial Natl. Bk. Bldg., OALR

MAINE

AUGUSTA

TURNER, O. W.....Augusta Trust Bldg., OALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
GILBERT, F. Y.....148 Park St., Op.AR
HASKELL, A. D.....145 High St., Op.
HOLT, E. E., JR.....723 Congress St., Op.A
SPALDING, J. A.....627 Congress St., Op.A

MARYLAND

ANNAPOLIS

WELCH, R. S. G.....46 State St. Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....Professional Bldg., Op.
FRIEDENWALD, HARRY.....1212 Eutaw Pla., Op.A
FRIEDENWALD, JONAS S.....1212 Eutaw Pl., Op.
GOLDBACK, L. J.....1012 N. Charles St., OALR
KEMLER, JOSEPH J.....1908 Eutaw Pl., OALR
LOOPER, EDWARD A.....605 Park Ave., OALR
PETERMAN, H. E.....518 N. Charles St., OALR
TARUN, WILLIAM.....605 Park Ave., Op.A
WODDS, HIRAM.....842 Park Ave., Op.A

CUMBERLAND

JONES, E. L.....First National Bank Bldg., OALR
ROBINSON, H. T.....Medical Bldg., OALR
SHARRETT, GEORGE O.....119 Bedford St., OALR

MASSACHUSETTS

BOSTON

BRYANT, A. G.....502 Beacon St., ALR
CARVILL, L. MAUD.....82 Commonwealth Ave., Op.
CHENERY, W. E.....377 Commonwealth Ave., ALR
CORBETT, JEREMIAH J.....520 Beacon St., OALR
DERBY, G. S.....23 Bay State Road, Op.
FREEDMAN, L. M.....419 Boylston St., ALR
GREENWOOD, ALLEN.....82 Commonwealth Ave., Op.
HURLEY, E. D.....419 Boylston St., Op.
HURLEY, E. H.....419 Boylston St., Op.
INGLIS, H. J.....Bay State Bldg., ALR
LANCASTER, W. B.....529 Commonwealth Ave., Op.
LEWIS, J. P.....543 Boylston St., ALR
MOSHER, H. P.....828 Beacon St., ALR
RICE, G. B.....270 Commonwealth Ave., ALR
RICHARDS, LYMAN G.....270 Commonwealth Ave., ALR
ROWLAND, WILLIAM D.....220 Clarendon St., Op.
SHANAHAN, T. J.....419 Boylston St., ALR
STANDISH, MYLES.....51 Hereford St., Back Bay, Op.
THOMPSON, P. H.....308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE.....9 Massachusetts Ave., Op.
TOBEY, G. L., JR.....270 Commonwealth Ave., ALR
WELLS, D. W.....The Westminster, Op.
WHITE, L. E.....390 Commonwealth Ave., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

JESSAMAN, L. W.....132 Union Ave., OALR

GARDNER

BONE, H. D.....29 Parker St., OALR

HOLYOKE

HUSSEY, E. J.....276 High St., Op.LR

LAWRENCE

CONLON, F. A.....Bay State Bldg., OALR
MERRILL, W. H.....Bay State Bldg., OALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTH ADAMS

THOMPSON, G. H.....18 Ashland St., OALR

NORTHAMPTON

COLLINS, J. D.....90 Main St., ALR
DOW, F. E.....4 West St., Op.

PALMER

MOORE, G. A.....Bank Bldg., OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
IRWIN, V. J.....389 Main St., OALR
IRWIN, V. J., JR.....274 Main St., OALR

TAUNTON

BAKER, H. B.....59 Main St., OALR

WESTFIELD

CLARK, F. T.....52 Broad St., OALR

WORCESTER

CAHILL, J. W.....390 Main St., OALR
CROSS, A. E.....Slater Bldg., Op.
ESTABROOK, CHARLES T.....390 Main St., OALR
MESSIER, A. EUGENE.....Park Bldg., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ANN ARBOR

FURSTENBERG, ALBERT C.....330 S. State St., ALR
SLOCUM, GEORGE.....328 E. Huron St., Op.

BATTLE CREEK

COLVER, B. N.....182 Manchester St., OALR
FRAZER, ROBERT H.....Battle Creek Sanitarium, ALR
HAUGHEY, WILFRID.....24 W. Main St., OALR
SLEIGHT, R. D.....Post Bldg., Op.
STEGMAN, L. V.....Sanitarium, Op.

BAY CITY

BAKER, C. H.....Crapo Block, OALR

DETROIT

AMBERG, EMIL.....David Whitney Bldg., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BENTLEY, NEIL.....1161 David Whitney Bldg., OALR

BERNSTEIN, E. J.....Professional Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....62 Adams St., W., ALR
FOWLER, WILLIAM.....Empire Bldg., OALR
GOUX, R. S.....545 David Whitney Bldg., OALR
HARREL, VOSS.....Holman Bldg.
HEATH, PARKER.....David Whitney Bldg., Op.
MCCLELLAND, CARL C.....David Whitney Bldg., OALR
MAIRE, L. E.....Kresge Bldg., Op.A
MERCER, R. E.....David Whitney Bldg., LR
MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, JOHN B.....90 E. Warren Ave., OALR
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....David Whitney Bldg., ALR
RENAUD, G. L.....Fine Arts Bldg., OALR
RYERSON, FRANK L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, B. R.....Shurly Bldg., ALR
SIMPSON, H. L.....David Whitney Bldg., ALR
WALKER, C. C.....Woodward at Warren, Op.
WENDEL, JACOB S.....David Whitney Bldg., ALR

ESCANABA

BOYCE, WILLIAM B.....1019 Ludington St., OALR

GRAND RAPIDS

DEAN, ALFRED.....304 Gilbert Blk. Bldg., Op.
GRANT, P. T.....Grand Rapids Natl. Bk. Bldg., OALR
ROBERTSON, F. D.....Ashton Bldg., OALR
ROLLER, L. A.....122 Monroe St., OALR
WELSH, D. E.....Powers Theater Bldg., OALR

IONIA

SEDGWICK, O. W.....Box 494, OALR

IRON MOUNTAIN

BOYCE, G. H.....408 E. D. St., OALR

JACKSON

WINTER, G. E.....44 Sun Bldg., OALR

KALAMAZOO

FULKERSON, C. B.....Kalamazoo National Bank Bldg., OALR
GRANT, F. E.....Kalamazoo National Bank Bldg., OALR
WILBUR, E. P.....Kalamazoo National Bank Bldg., OALR

LANSING

BURDICK, AUSTIN F.....416 Tussing Blk., OALR

MENOMINEE

ELWOOD, CALVIN R.....Spies Bldg., OALR

MT. CLEMENS

CROMAN, JOSEPH M., JR.....Washington Apts., OALR

ST. JOSEPH

MCDERMOTT, JOHN J.....St. Joseph Sanitarium, OALR

SAGINAW

MCKINNEY, ALEXANDER R.....Bearinger Bldg., OALR
ROGERS, A. S.....105 S. Jefferson Ave., OALR
WATSON, ROY S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

HOLDSWORTH, FRANK.....206 Wilhelm Blk., OALR

MINNESOTA

DULUTH

KNAPP, FRANK N.....309 Bradley Bldg., OALR
SHASTID, T. H.....Lyceum Bldg., Op.
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
TURNBULL, F. M.....Providence Bldg., OALR
WINTER, JOHN A.....Fidelity Bldg., OALR

FERGUS FALLS

KITTLESON, THEODORE N.....101 Lincoln Ave., OALR

HIBBING

MORSMAN, L. W.....Congdon Bldg., OALR

MANKATO

JAMES, J. H.....National City Bank Bldg., OALR

MINNEAPOLIS

BROWN, E. J.....900 Nicollet Ave., OALR
CAMP, W. E.....Donaldson Bldg., OALR
CAMPBELL, ROBT. A.....La Salle Bldg., OALR
CLARK, H. S.....La Salle Bldg., OALR
DORGE, RICHARD I.....76 Seymour Ave., S. E., OALR
HANSEN, E. W.....Donaldson Bldg., OALR
LITCHFIELD, J. T.....Yeats Bldg., OALR
LOOMIS, E. A.....Donaldson Bldg., OALR
MACNIE, J. S.....74 S. 11th St., OALR
MATTHEWS, JUSTUS.....Metropolitan Bldg., LR
MORTON, HOWARD MCI.....Metropolitan Bk. Bldg., OALR
MURRAY, WM. R.....1009 Nicollet Ave., OALR
NEWHART, HORACE.....Donaldson Bldg., ALR
OBERG, C. M.....Syndicate Blk., OALR
PATTERSON, W. E.....74 S. 11th St., OALR
PHELPS, K. A.....La Salle Bldg., OALR

PRATT, F. J., JR.....Metropolitan Bldg., OALR
PRATT, J. A.....Metropolitan Bldg., OALR
REYNOLDS, J. S.....La Salle Bldg., OALR
SMITH, A. E.....Donaldson Bldg., OALR
STROUT, E. S.....74 S. 11th St., OALR
STROUT, G. E.....Donaldson Bldg., OALR
WOOD, DOUGLAS.....Donaldson Bldg., OALR
WRIGHT, C. D.....Metropolitan Bk. Bldg., Op.A

NEW ULM

REINEKE, G. F.....

ROCHESTER

ANDERSON, C. M.....Mayo Clinic,
BENEDICT, W. L.....Mayo Clinic, Op.
GIPNER, J. F.....Mayo Clinic,
HEMPSTEAD, BERT E.....Mayo Clinic, ALR
LILLIE, H. I.....Mayo Clinic, ALR
NEW, G. B.....Mayo Clinic, LR
PRANGEN, A. DE H.....Mayo Clinic, Op.
WAGENER, H. P.....Mayo Clinic,

ST. PAUL

BERRISFORD, PAUL D.....Lowry Bldg., OALR
BINGER, H. E.....Lowry Bldg., OALR
BOECKMAN, EGIL.....Lowry Bldg., OALR
BRAY, E. R.....1440 Lincoln Ave., OALR
BROWN, JOHN C.....Lowry Bldg., OALR
BURCH, F. E.....Hamm Bldg., Op.
CONNOR, CHAS. E.....Hamm Bldg., OALR
FOGARTY, CHAS. W.....1826 Summit Ave., OALR
FULTON, JOHN F.....102 Western St., OALR
HOWARD, W. H.....137 N. Fairview Ave., OALR
LARSEN, CARL E.....Lowry Bldg., OALR
LEWIS, WILLIAM W.....836 Lowry Bldg., OALR
MALONEY, T. J.....Lowry Bldg., OALR
NELSON, L. A.....Lowry Bldg., Op.A
ROTHSCHILD, H. J.....Lowry Bldg., OALR
SHELLMAN, J. L.....Lowry Bldg., OALR
WHEELER, MERRITT W.....Lowry Bldg., OALR
WOLD, KARL C.....7th and Wacouta Sts., OALR

MISSISSIPPI

OXFORD

GUYTON, B. S.....Carter Bldg., OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., OALR
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR

MISSOURI

AURORA

O'DELL, TIMOTHY.....112 A Madison Ave., OALR

CARTHAGE

POST, WINFRED B.....First National Bank Bldg., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

NOYES, G. L.....Exchange Bk. Bldg., OALR
SNEED, C. M.....506 W. Broadway, OALR

KANSAS CITY

ALLEN, CHAS. E.....Waldheim Bldg., OALR
ALTRINGER, ARTHUR N.....Rialto Bldg., ALR
BEAL, HOMER A.....1002 Argyle Bldg.
BEIL, J. W.....Argyle Bldg., OALR
BELLOWS, GEORGE E.....Rialto Bldg., Op.
CURDY, R. J.....Rialto Bldg., Op.
CURRAN, EDWIN J.....Waldheim Bldg., Op.
DAVIS, HARRY B.....Lathrop Bldg., OALR
FOSTER, HAL.....Chambers Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Waldheim Bldg., OALR
KIMBERLIN, J. W.....Rialto Bldg., Op.
LANING, J. H.....Lathrop Bldg., ALR
LEA, J. A.....Rialto Bldg., ALR
LEMOINE, A. N.....Lathrop Bldg., Op.
LEONARD, A. C.....Bryant Bldg., OALR
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN.....Commerce Bldg., ALR
LUX, PAUL.....Commerce Bldg., ALR
MCALESTER, A. W., JR.....Bryant Bldg., Op.
MILLER, HUGH.....Chambers Bldg., OALR
MOSS, H. S.....Rialto Bldg., OALR
MOTT, J. S.....Argyle Bldg., OALR
MYERS, J. L.....Shukert Bldg., OALR
PAINTER, A. M.....Bryant Bldg., ALR
PATTERSON, J. M.....Bryant Bldg., OALR
ROBERTS, S. E.....Federal Reserve Bank Bldg., ALR
RUSSELL, E. L.....Argyle Bldg., OALR
SCHUTZ, W. H.....Bryant Bldg., OALR
SHOEMAKER, FERDINAND,
U. S. Vet. Hosp., No. 67, 11th and Harrison Sts., OALR
SHUMATE, D. L.....Shukert Bldg., OALR
THOMASON, H. E.....Rialto Bldg., OALR
TUREMAN, H. G.....Rialto Bldg., OALR
YAZEL, H. E.....Grand Ave. Temple, OALR

MOBERLY

DUTTON, C. K.....OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....Logan Bldg., OALR
KENNEY, WM. L.....Corby Forsee Bldg., OALR
MINTON, W. H.....Bartlett Bldg., OALR
PROUD, W. C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....537 Frisco Bldg., ALR
ARBUCKLE, M. F.....University Club Bldg., ALR
BALL, J. MOORES.....4500 Olive St., Op.
BARCK, CARL.....Humboldt Bldg., Op.A
BARDENHEIER, F. G. A.....Frisco Bldg., Op.A
BLACK, W. D.....Metropolitan Bldg., OALR
BURNS, S. S.....Humboldt Bldg., ALR
CHARLES, J. W.....Humboldt Bldg., Op.
COSTEN, J. B.....313 Wall Bldg., ALR
DONNELL, NEWMAN R.....Metropolitan Bldg., Op.
DYER, CLYDE P.....Metropolitan Bldg., Op.
EWING, ARTHUR E.....Metropolitan Bldg., Op.
GOLDSTEIN, M. A.....3858 Westminster Pl., ALR
GREEN, JOHN, JR.....Metropolitan Bldg., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HANSEL, F. K.....Missouri Bldg.
HARDESTY, J. F.....Metropolitan Bldg., Op.
HARDY, W. F.....Humboldt Bldg., Op.
HIGBEE, E. H.....Metropolitan Bldg., Op.
HOURN, G. E.....University Club Bldg., ALR
JACOBS, M. W.....Carleton Bldg., Op.
JENNINGS, J. E.....Carleton Bldg., OALR
LAMB, HARVEY.....Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LOEB, H. W.....1402 S. Grand Ave., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....Carleton Bldg., ALR
MCMAHON, B. J.....Missouri Theatre Bldg.
MYERS, E. L.....Wall Bldg.
PAYNE, RICHARD J.....University Club Bldg., ALR
PFINSGTEN, C. F.....508 N. Grand Ave., ALR
POST, LAWRENCE.....Metropolitan Bldg., Op.
POST, MARTIN H.....Metropolitan Bldg., Op.
PROETZ, ARTHUR W.....University Club Bldg., ALR
REIM, HUGO.....Metropolitan Bldg., Op.
SAUER, W. E.....Humboldt Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, F. A.....Metropolitan Bldg., Op.
SHAHAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....Carleton Bldg., Op.
SLUDER, GREENFIELD.....3542 Washington Ave., ALR
TOOKER, C. W.....Carleton Bldg., Op.

WESTLAKE, S. V.....Humboldt Bldg., ALR
WIENER, MEYER.....Carleton Bldg., Op.
WOODRUFF, F. E.....Metropolitan Bldg., Op.

SEDALIA

LOVE, J. G.....Ilgenfritz Bldg., OALR
TITSWORTH, GUY.....111 W. 4th St., Op.

SPRINGFIELD

BAILEY, H.....840 Landers Bldg., OALR
COFFELT, THEODORE A.....Woodruff Bldg., OALR
KLINGER, THOS. O.....Landers Bldg., OALR

MONTANA

ANACONDA

DUNLAP, LAWRENCE G.....Electric Light Bldg., OALR

BUTTE

DAYTON, G. O.....Lewisohn Bldg., OALR
DONOVAN, J. A.....Phoenix Bldg., OALR
MORSE, A. W.....Phoenix Bldg., OALR
POTTER, PETER.....Granate and Alaska Sts., OALR

GREAT FALLS

COULTER, C. F.....First Natl. Bank Bldg., OALR

LEWISTOWN

DAVIS, FRANK C..... ALR
PARSONS, J. G..... OALR

ROUNDUP

LEWIS, G. A.....29 Main St., OALR

NEBRASKA

BEATRICE

BAIRD, CHARLES G..... OALR
TUCKER, J. C.....114 S. 6th St., OALR

COLUMBUS

CAMPBELL, C. H.....Columbus State Bank Bldg., OALR

KEARNEY

FOX, CHAS. H.....2116 Central Ave., OALR

LINCOLN

BROOKS, E. B.....Terminal Bldg., OALR
CURTIS, W. L.....1503 S. 22nd St., OALR
DAYTON, W. L.....Funke Bldg., OALR
FURGASON, A. P.....Richards Block, OALR

HOMPES, J. J.....Security Mutual Bldg., OALR
SANDERSON, DAVID D.....Funke Bldg., OALR

NORFOLK

GADBOIS, A. E.....421 Norfolk Ave., OALR
NELSON, LLOYD L.....OALR

OMAHA

BANISTER, J. M.....Bankers Reserve Life Bldg., OALR
CALLFAS, W. F.....Brandeis Bldg., ALR
FAIRCHILD, NORA M.....Med. Arts Bldg., Op.
GIFFORD, HAROLD.....Med. Arts Bldg., Op.A
GIFFORD, SANFORD R.....Med. Arts Bldg., Op.
HANEY, W. P.....Med. Arts Bldg., OALR
ISAAC, DAVID.....Brandeis Theater Bldg., OALR
KNOVDE, A. R.....Omaha National Bank Bldg., ALR
KULLY, B. M.....631 City Natl. Bank Bldg., ALR
LEMERE, H. B.....Bankers Reserve Life Bldg., OALR
OWEN, F. S.....Med. Arts Bldg., OALR
PATTON, J. M.....Medical Arts Bldg., Op.
POTTER, GEORGE B.....402 City Natl. Bank Bldg., OALR
POTTS, J. B.....Med. Arts, ALR
RUBENDALL, CLARENCE.....Med. Arts Bldg., OALR
UREN, C. T.....537 City National Bank Bldg., OALR
WHERRY, W. P.....1500 Medical Arts Bldg., OALR

NEW HAMPSHIRE

NASHUA

NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

FISHER, JAMES A.....307 Edgemont Drive, OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. C.....1924 S. Illinois St., ALR
MCGIVERN, CHAS. S.....805 Pacific Ave., ALR

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

JERSEY CITY

PYLE, WALLACE.....18th and Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CHATTIN, J. F.....671 Broad St., Op.
HUGHES, LEE W.....1019 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....2 Lombardy St., OALR
O'CONNOR, F. O.....671 Broad St., OALR
ORTON, H. B.....671 Broad St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, E. S.....671 Broad St., OALR
ZEHNDER, A. CHARLES.....180 Fairmount Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

TRENTON

ADAMS, CHARLES.....52 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third St., ALR

WEST HOBOKEN

SACCO, A. G.....440 Clinton Ave., OALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....521 Luna Blvd., OALR

NEW YORK

ALBANY

BEDELL, A. J.....344 State St., Op.
BEGGS, W. F.....2 Lombardy St., OALR
DOWLING, J. I.....116 Washington Ave., OALR
HINMAN, EUGENE E.....146 Washington Ave., ALR
JUDGE, H. V.....46 Willett St., Op.

BINGHAMPTON

WATSON, H. D.....151 Front St., OALR

BROOKLYN

ALBRIGHT, LT. P. M., M. C. U. S. Navy, U. S. Naval Hosp., ALR
ARROWSMITH, HERBERT.....170 Clinton St., ALR
EVANS, JOHN N.....23 Schermerhorn St., Op.
GOLDING, JOSEPH E.....170 Hancock St., Op.
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, C. A.....17 Schermerhorn St.
KRUSKAL, ISAAC D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....186 Meserole St., Op.
MYERSON, MERVIN C.....198 Lincoln Pl., ALR

BUFFALO

ANDREWS, H. D.....23 Allen St., Op.
BENNETT, A. G.....26 Allen St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., OALR
BOZER, HERRMANN E.....438 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, DELZON N.....1001 Main St., ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, RAY.....560 Delaware Ave., Op.
FAIRBAIRN, JOHN F.....503 Delaware Ave., ALR
FLAGG, J. D.....473 Virginia Ave., Op.A
GLOSSER, HERBERT H.....448 Franklin St., OALR
HEALY, J. F.....503 Delaware Ave., ALR
HOWE, LUCIEN.....520 Delaware Ave., Op.
HUBBARD, A. E.....30 Radcliffe Rd., Op.
LEWIS, F. P.....454 Franklin St., Op.
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, W. L.....759 Richmond Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
SATTERLEE, R. H.....187 Delaware Ave., Op.
SERNOFFSKY, I.....37 Allen St., Op.
WEED, HARRY M.....196 Linwood Ave., Op.

CORTLAND

HIGGINS, REUBEN P.....20 Court St., OALR

DUNKIRK

JACKLE, A. F.....63 E. 4th St., Op.

GENEVA

SPENGLER, J. A.....423 S. Main St., OALR

GLENS FALL

FIELDING, FRED G.....72 Ridge St., ALR

ITHACA

BULL, E. L.....124 E. State St., OALR
KIRKENDALL, J. S.....315 N. Aurora, OALR

JAMESTOWN

HOTCHKISS, W. W.....195 Forest Ave., OALR
REGER, H. S.....641 Lakeview Ave., OALR

LIBERTY

DWORETSKY, JULIUS.....7 Law St., ALR

LOCKPORT

RINGUEBERG, EUGENE.....13 Main St., Op.

MT. VERNON

THOMSON, J. J.....3 Park Ave., OALR

NEW YORK CITY

ALMOUR, RALPH.....51 W. 73rd St.,ALR
AUERBACH, JULIUS.....120 W. 86th St., ALR
BALLIN, M. J.....108 E. 60th St., ALR
BELL, GEO. HUSTON.....40 E. 41st St., Op.
BERENS, CONRAD.....30 E. 40th St., Op.
BOONE, JAMES L.....N. Y. Eye and Ear Infir., ALR
BRUDER, JOSEPH.....230 W. 79th St., OALR
CARTER, W. W.....24 E. 48th St., ALR
COHEN, MARTIN.....1 W. 85th St., OALR
COHN, FELIX.....31 E. 60th St., ALR
COTT, C. C.....60 W. 88th St., ALR
DAVIS, A. E.....47 E. 57th St., Op.
DAVIS, G. E.....42 W. 77th St., OALR
DIXON, G. S.....40 E. 41st St., R
DUNNINGTON, JOHN H.....104 E. 40th St., Op.
FORBES, H. H.....40 E. 41st St., ALR
FREUDENTHAL, WOLF.....24 W. 88th St., ALR
FRIEDMAN, DAVID.....44 W. 77th St., ALR
GATEWOOD, W. L.....15 E. 48th St., ALR
GLOGAU, OTTO.....64 E. 91st St., OALR
GOLDMAN, HARRY G.....57 W. 58th St., ALR
GOTTLIEB, M. J.....814 West End Ave., ALR
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
GUTTMAN, J.....987 Madison Ave., OALR
HARTSHORNE, ISAAC.....20 W. 59th St.
HASKINS, W. H.....40 E. 41st St., ALR
HAYS, H. N.....22 W. 74th St., ALR
HETRICK, LLEWELLYN E.....20 E. 53rd St., ALR
HOPKINS, W. E.....515 Park Ave., OALR
HURD, L. M.....39 E. 50th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, SERGIUS.....418 Central Park, W., OALR
IRWIN, F. N.....114 E. 54th St., OALR
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....138 W. 86th St., OALR
JOHNSON, THOMAS H.....30 W. 59th St., Op.
JUDD, H. B.....47 E. 57th St., ALR
KERRISON, P. D.....58 W. 56th St., ALR
KEY, B. W.....100 W. 59th St., Op.
KING, J. J.....40 E. 41st St., ALR
KIRBY, DANIEL B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St.,Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, E. F.....12 W. 44th St., OALR
LA VIGNE, A. A.....11 E. 48th St., OALR
LUBMAN, MAX.....135 E. 65th St., ALR
MCCOY, JOHN.....750 Fifth Ave., ALR
MCDONALD, DENNIS.....17 E. 38th St.
MCDONALD, DENNIS J.....17 E. 38th St.

MAY, C. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....12 E. 86th St., ALR
MAYER, EMIL.....40 E. 41st St., ALR
MEIERHOF, E. L.....1140 Madison Ave., OALR
MINSKY, H.....215 W. 88th St.
MITTENDORF, A. D.....399 Park Ave., Op.
MITTENDORF, W. K.....115 E. 53rd St., Op.A
MORRISON, W. W.....39 E. 50th St.
NISSELSON, MAX.....616 Madison Ave., OALR
NORTHCOTT, T. A.....17 E. 38th St.
OPPENHEIMER, SEYMOUR.....45 E. 60th St., OALR
PALMER, ARTHUR.....2178 Broadway
PHILLIPS, W. C.....40 W. 47th St., ALR
REESE, R. G.....50 W. 52nd St., Op.
SCAL, J. C.....44 W. 74th St.
SCHILLER, A. N.....255 West End Ave., ALR
SCHLIVEK, KAUFMANN.....1000 Park Ave., Op.
SOMBERG, JOSEPH S.....60 E. 67th St., Op.
STARK, JESSE B.....270 Park Ave., Op.
STURGIS, LEIGH F.....128 W. 87th St., LR
WEEKS, JOHN E.....20 E. 53rd St., Op.
WEEKS, WEBB W.....20 E. 53rd St., Op.
WHEELER, J. M.....30 W. 59th St., Op.
WIENER, ALFRED.....550 Park Ave., OALR
YANKAUER, SIDNEY.....616 Madison Ave., ALR

NIAGARA FALLS

PRICE, N. W.....445 Third St., OALR
ROOKER, A. L.....225 Gluck Bldg., ALR

NYACK-ON-HUDSON

TOMS, S. W. S ..... ALR

OLEAN

TINDOLPH, LEA W.....First Natl. Bank Bldg., OALR

ONEIDA

CROCKETT, R. L..... Op.

POUGHKEEPSIE

KRIEGER, W. A.....35 Market St., OALR

ROCHESTER

BARBER, FRANK.....75 S. Fitzhugh St., Op.
CARROLL, G. G.....614 Main St.,W., OALR
CLARK, L. H.....37 Monroe Ave., OALR
JONES, LEONARD W.....53 S. Fitzhugh St., OALR
LERNER, MACY L.....332 Park Ave., Op.
MCDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....303 Alexander St., ALR
SHAPERO, I. M.....289 Alexander St., OALR
SNELL, A. C.....53 S. Fitzhugh St., Op.

SYRACUSE

BROWN, MORTIMER G.....802 University Bldg., ALR
BRITTON, G. S.....University Blk., OALR
BRUST, H. O.....720 S. Crouse Ave., ALR
FOWLER, S. R.....University Bldg., ALR
GILLETTE, DAVID F.....811 Park St., Op.
JOY, H. H.....232 Brattle Rd.
KAUFFMAN, ARNOLD B.....Gurney Bldg., ALR
KLINE, H. G.....120 E. Washington, ALR
MARLOW, FRANK W.....University Bldg., Op.

TROY

SULZMAN, F. M.....1831 Fifth Ave., OALR

UTICA

BEATTIE, W. HENRY.....252 Genesee St., OALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.

NORTH CAROLINA

ASHEVILLE

BRIGGS, H. H.....73 Haywood St., OALR
GREENE, JOSEPH B.....Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P.....511 Independence Bldg., OALR
PEELER, C. N.....W. 7th St., OALR
SLOAN, HENRY L.....Independence Bldg., Op.
WHISNANT, A. M.....Realty Bldg., OALR

GREENSBORO

REAVES, W. P.....Reaves Infirmary, OALR

NEW BERN

DANIELS, R. L.....Elks' Temple, OALR

RALEIGH

WRIGHT, J. B.....Citizens Natl. Bank Bldg., OALR

SALISBURY

BRAWLEY, R. V.....Wallace Bldg., OALR

WILMINGTON

MURPHY, J. G.....Murchison Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W.....O'Hanlon Bldg., OALR

NORTH DAKOTA

FARGO

RINDLAUB, ELIZABETH P.....De Lendrecie Blk., OALR
RINDLAUB, J. H.....De Lendrecie Blk., OALR
RINDLAUB, M. P., JR.....De Lendrecie Blk., OALR

MINOT

MCCANNELL, A. D.....Colcord Bldg., OALR

WAHPETON

RICE, C. P.....Sterns Bldg., OALR

OHIO

AKRON

BROWN, L. E.....1129 2nd Natl. Bank Bldg., ALR
MOORE, T. K.....Central Sav. & Trust Bldg., OALR
STEVENSON, D. W.....165 E. Market St., OALR

ALLIANCE

KING, G. L.....437 E. Market St., OAR

BELLEFONTAINE

BUTLER, ROBERT H.....227 W. Columbus Ave., OALR
HARBERT, J. P.....135 N. Ulain St., OALR

CANTON

SCHILD, E. H.....9th and Cleveland, Op.A

CINCINNATI

BROWN, A. L.....Union Central Bldg.
ASHBURY, MARY K.....707 Race St., Op.
BEYER, ARTHUR G.....19 Garfield Pl., OALR
GOODYEAR, HENRY M.....3029 Fairfield Ave., ALR
HANS, C. L.....19 W. 7th St., OALR
HARRIS, W. C.....Groton Bldg., ALR
IGLAUER, SAMUEL.....707 Race St., ALR
JONES, CHARLES C.....19 Garfield Pl., ALR
KING, C.....Union Central Bldg., Op.
KING, EDWARD.....707 Race St., ALR
LAMB, F. W.....209 Provident Bk. Bldg., OALR
MITHOEFER, WILLIAM.....19 Garfield Pl., ALR
MURPHY, W. E.....7th and Race Sts., OALR
PHINNEY, F. D.....22 W. 7th St., OALR
RAY, VICTOR.....19 W. 7th St., OALR
SIEGEL, F. X.....2600 Union Central Bldg., OALR
STANBERRY, HENRY.....Doctors Bldg., Op.AR
STEWART, T. M.....Union Trust Bldg., OALR
STRICKER, LOUIS.....Groton Bldg., Op.
TANGEMAN, H. F.....Union Central Bldg., ALR

THOMPSON, E. H.....Vindonissa Bldg., 19 W. 7th St., OALR
URNER, M. H.....Union Central Life Bldg., OALR
VAIL, D. T.....24 E. 8th St., Op.
VAIL, H. H.....24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J.....8314 Euclid Ave., OALR
BRUNER, ABRAM B.....Guardian Bldg., Op.
BRUNER, W. E.....Guardian Bldg., Op.
BURKE, T. A.....Rose Bldg., OALR
COGAN, J. E.....Rose Bldg., OALR
CUTLER, FRANKLIN E.....1025 Schofield Bldg., ALR
FORSYTHE, S. T.....235 Colonial Arcade, OALR
INGERSOLL, J. M.....Osborn Bldg., ALR
KENDALL, M. R.....Osborn Bldg., ALR
LARGE, S. H.....2323 Prospect Ave., ALR
LENKER, J. N.....Osborn Bldg., ALR
MCCALL, JULIUS W.....Osborn Bldg., ALR
MCDONALD, C. L.....Ainsfield Bldg., OALR
METZ, R. B.....Guardian Bldg., Op.
METZENBAUM, MYRON.....Rose Bldg., ALR
MONSON, S. H.....Ainsfield Bldg., OALR
MULLIN, W. V.....Cleveland Clinic, ALR
MUSSUN, WILLIAM G.....Osborn Bldg., ALR
NELSON, C. F.....Schofield Bldg., OALR
PITKIN, C. E.....Osborn Bldg., ALR
PRENDERGAST, D.....Hanna Bldg., OALR
QUITTNER, S. S.....3912 Prospect Ave., OALR
RIEGER, WALTER H.....Hanna Bldg., ALR
RUST, E. G.....Hanna Bldg., Op.A
SHACKLETON, W. E.....2323 Prospect Ave., Op.
SHIRAS, H. H.....Guardian Bldg., Op.
SHIREY, OSCAR M.....Osborn Bldg., ALR
SIMONDS, O. F.....Hanna Bldg., Op.
STOTTER, A. L.....1148 Euclid Ave., OALR
TRIPP, I. A.....Rose Bldg., OALR
TUCKERMAN, W. C.....Osborn Bldg., Op.
TUCKERMAN, W. H.....Osborn Bldg., OALR
WAUGH, J. M...Cleveland Clinic, Euclid Ave. at 93rd St., ALR

COLUMBUS

BEATTY, H. G.....150 E. Broad St., OALR
BROWN, J. E.....370 E. Town St., OALR
CLARK, I. G.....188 E. State St., OALR
HAUER, A. M.....327 E. State St., OALR
PROUT, A. W.....370 E. Town St., OALR
SILBERNAGEL, C. E.....283 E. State St., OALR
SULZER, G. A.....Medical Arts Bldg., OALR
THOMAS, F. W.....74 S. 5th St., OALR
TIMBERMAN, ANDREW.....112 E. Broad St., OALR
WRIGHT, J. W.....Central National Bank Bldg., Op.

DAYTON

BINKLEY, R. S.....Fidelity Med. Bldg., OALR
BLACKBURN, W. J.....Reibold Bldg., OALR
DUTROW, H. V.....Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, PERLEY H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....58 Cambridge Ave., OALR

ELYRIA

GILL, GEORGE.....Elyria Sav. & Trust Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts.

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....424 Broadway, OALR

MANSFIELD

GARBER, J. M.....48 Park Ave., OALR
WISE, R. C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....231 Third Ave., OALR

MARION

BRICKLEY, D. W.....196 W. Center St., OALR

MASSILLON

SCOTT, M. E.....1st Natl. Bk. Bldg.

MIDDLETOWN

WILLIAMS, WALTER H.....Castell Bldg., OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SANDUSKY

BLISS, C. B.....411 Columbus Ave., OALR

SPRINGFIELD

EASTON, J. C.....Fairbanks Bldg., OALR
HARTLEY, F. A.....Fairbanks Bldg., OALR
HOGUE, D. W.....Fairbanks Bldg., OALR
MINOR, C. L.....Fairbanks Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, W. W.....Nasby Bldg., OALR
ALTER, F. W.....Toledo Medical Bldg., OALR
DENMAN, IRA O.....Ohio Bldg., OALR
KING, CHARLES R.....Spitzer Bldg., OALR
LUKENS, CHARLES.....316 Michigan Ave., OALR
SNYDER, W. H.....212 Ontario St., OALR
STEINFELD, A. L.....125 15th St., OALR

WARREN

STIMSON, GEORGE W.....Packard Apt., ALR

XENIA

MADDEN, REID.....Allen Bldg., OALR
SHIELDS, L.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD J.....Dollar Bldg., OALR
GIBSON, R. D.....Dollar Savings and Trust Bldg., OALR
HARTZELL, S. M.....Dollar Bank Bldg., OALR

ZANESVILLE

CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Central Natl. Bank Bldg., OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Oklahoma Ave., OALR

MCALESTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....706 1st Natl. Bank Bldg., OALR
FERGUSON, E. S.....902 Medical Arts Bldg., OALR
GUTHRIE, A. L.....Amer. Natl. Bank Bldg., OALR
MCHENRY, D. D.....Med. Arts Bldg., OALR
NEWTON, L. A.....Med. Arts Bldg., OALR

TULSA

BRASWELL, C. J.....726 Mayo Bldg., OALR
COOK, W. ALBERT.....506 Palace Bldg., OALR
ROTH, A. W.....Palace Bldg., OALR

OREGON

PORTLAND

AINSLIE, GEORGE.....Oregonian Bldg., OALR
BOUVY, HARRY M.....800 Pittock Blk., OALR
BRUERE, G. E.....Journal Bldg., OALR
DAVIS, RALPH F.....Selling Bldg., OALR
FENTON, RALPH A.....806 Medical Arts Bldg., OALR
HENTON, GEORGE EARL.....Morgan Bldg., OALR
JOHNSTON, WILSON.....309 Albemarle Terrace, OALR
KIEHLE, F. A.....Corbett Bldg., OALR
KISTNER, F. B.....Stevens Bldg., ALR
MCCOOL, JOSEPH L.....725 Sherwood Drive, Op.

PENNSYLVANIA

ALLENTOWN

HARDING, F. B.....22 S. 10th St.

ALTOONA

GLOVER, S. P.....1118 12th Ave., OALR

BEAVER

SCROGGS, J. J.....218 E. 3rd St., OALR

BETHLEHEM

CHASE, WALTER D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....Reiber Bldg., OALR
HAZLETT, L. R.....117 S. Main St., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR
STAHLMAN, F. C.....318 Fifth St., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.

CYNWYD

TUCKER, GABRIEL E.....107 Lodges Lane, ALR

ERIE

DENNIS, D. N.....221 W. 9th St., Op.A
DENNIS, E. P.....311 W. 10 St., OALR
DUNN, I. J.....Masonic Temple, OALR
FUST, J. H.....138 W. 9th St., OALR
HEARD, C. F.....Masonic Temple, OALR
JACKSON, J. D.....2324 W. 8th St.
RUSSELL, J. A.....206 Masonic Temple, ALR

SCHLINDWEIN, G. W.....138 W. 9th St., OALR
SHREVE, O. M.....162 W. 8th St., OALR

FRANKLIN

JOBSON, G. B.....Printz Bldg., OALR

GREENSBURG

FAIRING, JOHN W.....204 Tribune Bldg., OALR
MCKEE, C. W.....Coulter Bldg., OALR

HARRISBURG

DAILY, G. L.....713 N. 3rd St., OALR
FARNSLER, H. H.....1438 Market St., OALR
GRACEY, GEORGE F.....209 State St., OALR
PARK, J. W.....32 N. 2d St., OALR
REBUCK, C. S.....412 N. 3d St., OALR

HAZLETON

REICHE, O. C.....328 W. Broad St., OALR

HOMESTEAD

BROWN, W. E.....8th Ave. and Amity St., OALR

HUNTINGDON

SEARS, W. H.....514 Penn St., OALR

JOHNSTOWN

BARKER, O. G. A.....Johnstown Trust Bldg., Op.
HARRIS, CLARENCE M.....Johnstown Trust Bldg., OALR
RUSH, CALVIN C.....342 Main St., OALR

MIDDLETOWN

GEORGE, H. W.....19 N. Union St., Op.

NEW KENSINGTON

MCCONNELL, T. E.....Alter Bldg., OALR
STEIM, JOSEPH M.....1st Natl. Bk. Bldg., Op.

NORTH EAST

HEARD, MARY K.....41 W. Main St., Op.

PHILADELPHIA

ANNON, WALTER T.....259 S. 17th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....2117 Chestnut St., ALR
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BROWN, SAMUEL HORTON.....1901 Mt. Vernon St., Op.
BRUMM, SETH A.....Stock Exchange Bldg., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, M. M.....2045 Chestnut St., Op.
CLERF, LOUIS H.....128 S. 10th St., ALR
COATES, G. M.....1736 Pine St., ALR

COHEN, SAMUEL.....1626 Spruce St., ALR
CREIGHTON, W. J.....258 S. 18th St., Op.
CROSKEY, J. W.....Aldine Hotel, Op.
DAVIS, J. L.....135 S. 18th St., ALR
DEICHLER, LYN WALTER.....2028 Chestnut St., OALR
DINTENFASS, HENRY.....1714 Pine St., ALR
ERSNER, M. S.....1729 Pine St., ALR
FELT, C. L.....N. E. Cor. Chestnut & 20th Sts., ALR
FOX, L. W.....303 S. 17th St., Op.
GLEASON, E. B.....2033 Chestnut St., ALR
GODDARD, H. M.....1531 Spruce St., ALR
GRISCOM, J. M.....1925 Chestnut St., Op.
HAWMAN, E. G.....4051 N. Broad St., OALR
HEED, C. R.....1205 Spruce St., Op.
HERBERT, A. W.....119 E. Lehigh Ave., OALR
HOLLOWAY, T. B.....1819 Chestnut St., Op.
HUSIK, DAVID N.....1610 Spruce St., ALR
JACKSON, C.....128 S. 19th St., L
KAUFMAN, A. S.....1923 Spruce St., ALR
KEELER, J. C.....254 S. 16th St., ALR
KRAUSS, FREDERICK.....1701 Chestnut St., OALR
LAESSLE, H. A.....The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A.....Medical Arts Bldg., Op.
LEWIS, FIELDING O.....259 S. 17th St., ALR
LOFTUS, J. E.....605 Medical Arts Bldg., ALR
LUKENS, R. McD.....1308 Hunting Park Ave., ALR
MACFARLAN, DOUGLAS.....1805 Chestnut St., OALR
MACKENZIE, ALICE V.....1724 Spruce St., OALR
MACKENZIE, GEORGE W.....1724 Spruce St., ALR
MILLER, E. B.....2008 Walnut St., Op.
MITCHELL, E. K.....704 W. Lehigh Ave., ALR
MOORE, W. F.....255 S. 16th St., ALR
OLSHO, SYDNEY L.....235 S. 15th St., OALR
O'REILLY, C. A.....Medical Arts Bldg., ALR
PETER, LUTHER C.....N. E. Cor. Chestnut and 20th Sts., Op.
PONTIUS, P. J.....1831 Chestnut St., OALR
POSEY, WM. CAMPBELL.....21st and Chestnut Sts., Op.
RADCLIFFE, MCCLUNEY.....1906 Chestnut St., Op.
REESE, M. D.....3527 Hamilton St.
REESE, WARREN S.....230 S. 21st St., Op.
RIDPATH, R. F.....1928 Chestnut St., ALR
ROMMELL, J. C.....4501 N. Broad St., OALR
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLET, HUNTER W.....230 S. 21st St., Op.
SCHNEIDEMAN, T. B.....Professional Bldg., Op.
SCHWEINITZ, G. E. DE.....1705 Walnut St., Op.
SCHWENK, P. N. K.....1417 N. Broad St., Op.
SENER, W. J.....130 S. 18th St., OALR
SHEMELEY, W. G., JR.....1724 Spruce St., OALR
SKILLERN, R. H.....1811 Spruce St., LR
SKILLERN, SAMUEL R., JR.....2017 Walnut St., ALR

SMITH, S. McC.....1429 Spruce St., ALR
SPENCER, W.....1830 S. Rittenhouse Sq., OALR
STILES, C. M.....1831 Chestnut St., Op.
STOUT, PHILIP S.....Medical Arts Bldg., ALR
STROUSE, FRED M.....1301 Spruce St., ALR
SWEET, W. M.....1205 Spruce St., Op.
WAGERS, A. J.....The Lenox, 13th and Spruce Sts., ALR
WHELAN, GEORGE L.....257 S. 16th St., ALR
WILLIAMS, H. J.....5908 Green St., Germantown, ALR
ZENTMAYER, WILLIAM.....1506 Spruce St., Op.

PITTSBURGH

BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....Diamond Bank Bldg., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CURRY, G. E.....Westinghouse Bldg., Op.
DICKINSON, B. M.....5634 Stanton Ave., E. Liberty Sta., ALR
FISCHER, NATHANIEL A.....Union Trust Bldg., ALR
GROSS, JULIUS E.....Jenkins Arcade Bldg., ALR
GUERINOT, A. J.....Westinghouse Bldg., OALR
HAGEMAN, JOHN A.....Highland Bldg., ALR
HECKEL, E. B.....Jenkins Arcade Bldg., Op.
KNEEDLER, G. C.....Professional Bldg., ALR
KREBS, A.....Jenkins Arcade Bldg., Op.
LINN, J. G.....Jenkins Arcade Bldg., OALR
McCREADY, J. H.....Empire Bldg., ALR
McKEE, GEORGE J.....1118 Westinghouse Bldg., ALR
MacLACHLAN, A. A.....May Bldg., ALR
MARKEL, J. C.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....Diamond Bank Bldg., ALR
MEANOR, W. C.....Westinghouse Bldg., Op.
MURDOCH, J. F.....Bessemer Bldg., Op.
PATTERSON, E. J.....Westinghouse Bldg., ALR
SEEGMAN, SIMON.....Jenkins Arcade Bldg., ALR
SHUMAN, G. H.....Park Bldg., Op.
SMITH, S. S.....Jenkins Arcade Bldg., Op.
STIEREN, EDWARD.....Union Trust Bldg., Op.
STURM, S. A.....Jenkins Arcade Bldg., Op.
TAYLOR, MARTIN C.....1206 May Bldg., Op.
TODD, G. C.....307 Westinghouse Bldg., OALR
TURNER, HUNTER H.....902 Professional Bldg., Op.
VAN KIRK, V. E.....Union Arcade Bldg., Op.
WEIMER, EDGAR S.....332 Lehigh Ave., Op.
WEISSER, E. A.....Empire Bldg., Op.

POTTSVILLE

MOORE, G. H.....Schuylkill Bldg., OALR

READING

STOCKLER, J. A.....332 N. 5th St., OALR

RIDGEWAY

McALISTER, J. C.....10 S. Mill Ave., OALR

SCRANTON

CORSER, J. B.....345 Wyoming Ave., Op.
CURTIN, EUGENE A.....Connell Bldg., OALR
FREY, C. L.....529 Vine St., OALR
SULLIVAN, JOHN J., JR.....Traders Bank Bldg., ALR

SHARON

NORTON, ROY R.....107 B. E. State St., OALR

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BECKER, H. M.....49 S. 4th St., OALR

UPPER DARBY

BOEHRINGER, H. W.....1014 Cooperstown Rd., Op.
SHANNON, C. E.....104 Copley Rd., Op.

WARREN

BALL, M. V.....214 Penn Ave., W., OALR

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MCKENNAN, J. W.....Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WILKES-BARRE

BUCKMAN, E. V.....70 S. Franklin St., OALR
CARR, G. W.....30 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.A
MCLAUGHLIN, P. A.....73 S. Washington St., OALR
SCHAPPERT, N. L.....59 S. Washington St., OALR
TAYLOR, LEWIS H.....83 S. Franklin St., OALR

YORK

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ADAMS, F. M.....122 Waterman St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....114 Waterman St., OALR
LEECH, J. W.....369 Broad St., OALR
MESSINGER, H. C.....170 Broad St., Op.
MUNCY, W. M.....25 Waterman St., OALR
PORTER, L. B.....195 Thayer St., OALR

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth, OALR
TOWNSEND, JOHN F 85 Wentworth, Cor. St. Phillips St., OALR

GREENVILLE

CARPENTER, E. W.....201 E. North St., ALR
MAULDIN, L. O.....Wallace Bldg., OALR

SOUTH DAKOTA

ABERDEEN

ALWAY, ROBERT D.....423 S. Lincoln St., OALR

HURON

GROSVENOR, L. N.....378 Dakota St., OALR

MITCHELL

BOBB, E. V.....First Natl. Bank Bldg., OALR

RAPID CITY

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GREGG, J. B.....Security Natl. Bank Bldg., OALR
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JOHNSON, A. E.....Citizens Bank Bldg., OALR

YANKTON

HOHF, JULIUS.....Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J.....630 Volunteer Bldg., OALR
HOGSHEAD, J. M.....602 Georgia Ave., OALR

DYERSBURG

BREWER, JOHN D.....618 Main St., OALR

JACKSON

DANCY, A. B.....First National Bank Bldg., OALR

KNOXVILLE

KINCAID, J. H.....421 W. Church Ave., OALR
REAVES, CHAS. R.....Box No. 187, OALR

MEMPHIS

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BLUE, J. B.....Exchange Bldg., OALR

ELLETT, E. C.....Exchange Bldg., Op.
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WATKINS, E. D.....Exchange Bldg.

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WOOD, HILLIARD.....Independence Bldg., OALR

TEXAS

AUSTIN

BOERNER, MORRIS H.....Scarborough Bldg., OALR

BROWNWOOD

ANDERSON, W. B.....507 Main Ave., OALR

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JOHNSON, O. H.....U. S. Veterans Bureau, OALR
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SEAY, D. E.....Dallas Co. State Bank Bldg., OALR
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STROUD, E. F .....Med. Arts Bldg.

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FULLER, T. E.....803 Pine St., OALR
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TYLER

VAUGHN, E. H.....113½ W. Ferguson St., OALR

WACO

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OGDEN

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ONTARIO

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MCKELVEY, A. D. M.....160 Bloor St., ALR
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MONTREAL

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LASSALLE, ALBERT.....493 St. Denis St., OALR
MACMILLAN, JOHN A.....129 Stanley St., Op.
MCKEE, S. HANFORD.....158 Crescent St., Op.
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WHITE, E. H.....756 Sherbrook St., ALR

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PRAGUE

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GRAY, A. A.....

SWITZERLAND

ZURICH

BARKAN, A.....Credit Swisse, OALR
NAGER, PROF. F.....Freiestrasse 20

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